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AUGUST 1953

Postgraduate Medicine

Official Journal
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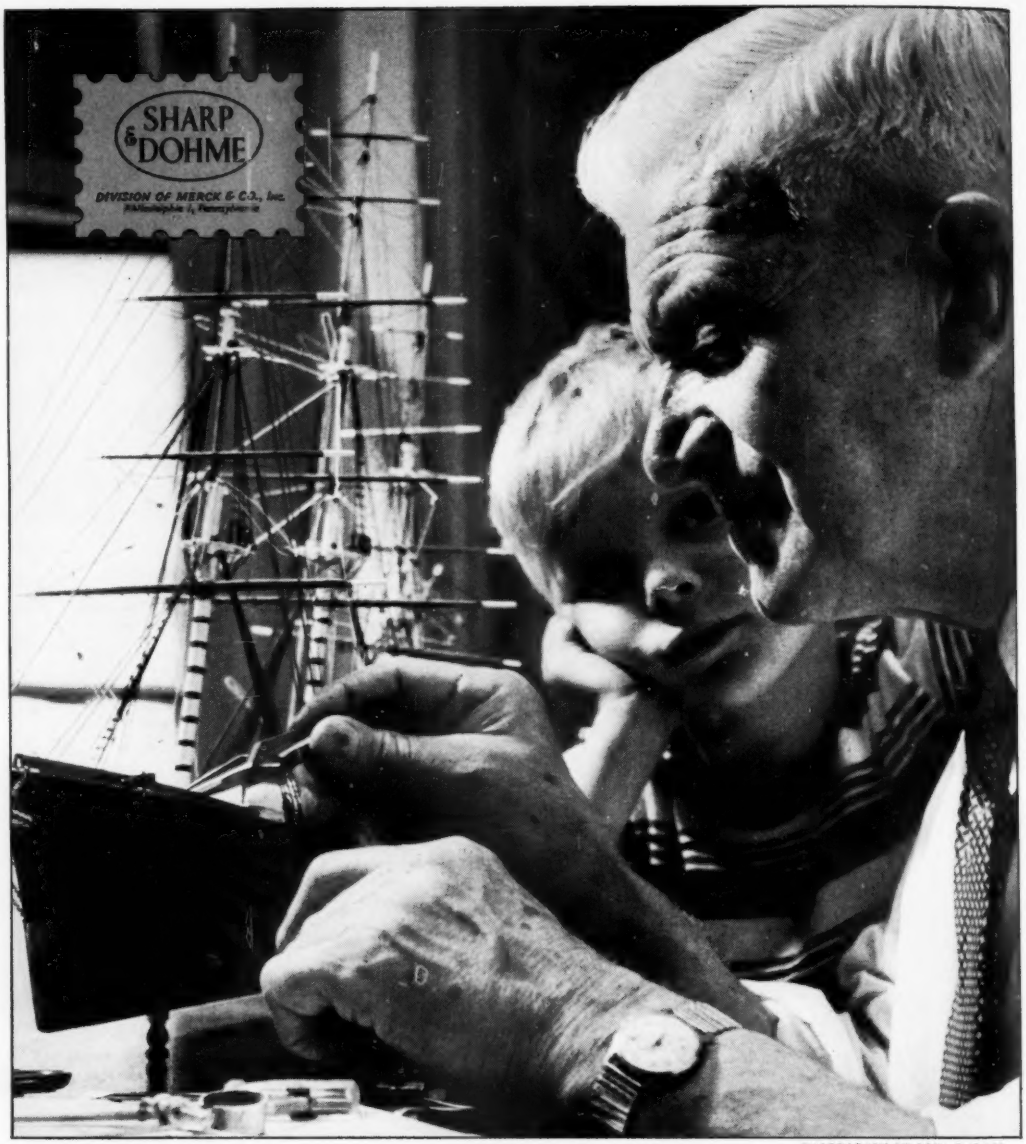

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Postgraduate Medicine

Volume 14 · Number 2
AUGUST · 1953

OFFICIAL JOURNAL OF THE INTERSTATE POSTGRADUATE MEDICAL ASSOCIATION

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Sexual Behavior in the Human Female

This book is a scientific study of sexual behavior in American women of various ages, educational levels, religious adherences, parental backgrounds. The material is based on data accumulated over the past 15 years in personal interviews with almost 8000 women; in research carried on in various areas of science; and in an exhaustive study of the literature in many fields.

The authors present data on the *incidence and frequency* with which women participate in various types of sexual activity. Factors of age and background which modify the type and extent of activity are clearly indicated. Special significance of each type of behavior (including the woman's own reaction to it) is measured wherever the data allow such a measurement. Summarized comparisons with male behavior are made throughout.

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Without question, the book will prove tremendously useful to the physician in the management of the many problems of sexual maladjustment which he sees among his female patients.

By the Staff of the Institute for Sex Research at Indiana University. Alfred C. Kinsey, Wardell B. Pomeroy, Clyde E. Martin, Paul H. Gebhard, Research Associates; and Jean M. Brown, Cornelia V. Christenson, Dorothy Collins, Ritchie G. Davis, William Dellenback, Alice W. Field, Hedwig G. Leser, Henry H. Remak, Eleanor L. Roehr. Consultants for all or for specialized parts of the book: Jerome Cornfield, Harold Dorn, Robert Laidlaw, Karl Lashley, Emily Mudd. 842 pages, 6" x 9", with 151 charts; 179 tables; 4 illustrations. $8.00.

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Your Questions answered

Readers may send questions from their own practice or other medical problems which will be answered by qualified consultants.

• DEAFNESS FOLLOWING USE OF DIHYDROSTREPTOMYCIN

Question: I had pulmonary tuberculosis, and after receiving about 250 gm. of dihydrostreptomycin and 770 gm. of PAS within a period of 8 months, I lost my hearing. These medicines were discontinued in July 1951, with the first appearance of tinnitus and slight impairment of hearing. In spite of the absence of these drugs, my deafness has progressed gradually so that at present I am more or less completely deaf. We could not locate any other probable cause of the deafness. Other ENT examinations give normal findings, and show nothing of importance. The last audiogram revealed 98.9 per cent hearing loss in the left ear and 97.6 per cent in the right. No audiometric examination could be performed recently due to lack of facilities in the place where I am staying. Treatment so far received, but without any beneficial effects, consists of: thiamine, B complex preparations, vitamin A, vitamin B_{12}, PROSTIGMIN®, strychnine. Cortisone and ACTH had not been given any trial because of their contraindications to my pulmonary lesion. I tried to use a hearing aid, but it did not help me.

As to my pulmonary disease, it appears to be under control, with marked improvement as shown radiologically. Present treatment consists of NYDRAZID® and artificial pneumoperitoneum. I have never suffered from vertigo or any other signs referable to vestibular dysfunction, although the tinnitus now is very annoying. Any recommendations or suggestions to regain my normal hearing will be greatly appreciated.

M.D.—Philippines

ANSWER: Dihydrostreptomycin in the dosage and duration of treatment reported, averaging at least 1 gm. daily for 8 months, is prone to produce serious auditory impairment. It is not unusual for the deafness to progress and even to first make its appearance after medication is discontinued. Dihydrostreptomycin is more toxic to the auditory apparatus than streptomycin while the reverse is true as regards vestibular toxicity. The incidence of toxic and allergic reactions to both dihydrostreptomycin and streptomycin is reduced by intermittent use, such as 1 gm. every third day or twice a week, with little, if any, loss in effectiveness. It is wise to have audiometric studies made prior to prolonged treatment with streptomycin or dihydrostreptomycin and audiograms should be rechecked frequently during treatment.

I note the questioner reports he is taking nydrazid (isoniazid) alone. Since bacterial resistance to isoniazid, when taken alone, develops rapidly, it might be advisable to add daily PAS.

Unfortunately, auditory impairment (nerve deafness) due to the streptomycin drugs appears to be irreversible.

SUMNER S. COHEN

• BORIC ACID FOR NASAL CONDITIONS

Question: In the treatment of nasal and sinus conditions I find a near-saturated solution of boric acid very useful, with no undesirable side effects. Are there any contraindications to the use of this solution in nasal conditions? I also add PHEMEROL CHLORIDE® to this solution to make 1:5000. Do you see any reason to the contrary?

M.D.—Tennessee

ANSWER: The use of a near-saturated solution of boric acid in the treatment of nasal and sinus conditions has no apparent rational basis. It should be tested for its effect on ciliary motility and on the nasal mucous membrane, as well as for its effect on nasal *p*H, in order to establish a clearer understanding of the manner in which the drug may act.

NOAH D. FABRICANT

(Continued on page A-14)

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• X-RAY THERAPY FOR BRAIN TUMOR

Question: On April 6, 1953, a 56 year old married woman ur.derwent a right temporal lobectomy because of brain tumor. Her family is very anxious to know what can be done in the way of x-ray therapy as a follow-up, inasmuch as the operating surgeon informed them that the tumor was moderately malignant. What would you recommend in the way of x-ray therapy, and how often?

M.D.—Michigan

ANSWER: While an answer to this question would depend entirely on the type of tumor involved, Dr. George Ehni, our associate neurosurgeon, has commented as follows: "There are three types of tumor occurring in the location in question which are moderately responsive to x-ray therapy. These are ependymoblastoma, glioblastoma multiforme and hemangioblastoma. The tumor dose is generally considered to be between 3000 r and 4000 r (the brain itself will stand a maximum of 6000 r in divided doses), and is purely palliative. I am unfamiliar with any claims of cure having been effected by x-ray therapy in tumors of these types. If the tumor involved is an astrocytoma or an oligodendroglioma, both of which are nonencapsulated, x-ray therapy is contraindicated."

Dr. Gilbert H. Fletcher, our radiologist, concurs in Dr. Ehni's comments.

R. LEE CLARK, JR.

• BRONCHIAL ASTHMA

Question: A patient of mine, a 46 year old woman, has had bronchial asthma for 8 years. She is sensitive to house dust, feathers, and dog and horse hair, and also has a drug sensitivity to quinine derivatives and phenolphthalein. About 8 months ago severe generalized urticaria developed which was resistant to all treatments (ADRENALIN®, antihistamines and calcium). Cortisone and ACTH bring it under control, but on discontinuance of these drugs the urticaria reappears within 10 days. This patient is very nervous, highly sensitive, a perfectionist, and has a serious emotional problem which seems to be concentrated about her relationship to her 17 year old son.

I would appreciate your advice concerning this case, including the advisability of psychiatric or psychoanalytic treatment. I would also like your opinion of the new drug, TYRAL®, which is supposed to be a precursor of epinephrine.

M.D.—California

ANSWER: Competent and adequate allergic supervision over a protracted period is the first necessity in this difficult case. If this has been done, I would suggest psychiatric consultation to take care of the psychic problem which seems to me to be the predominant cause at present.

Regarding the new drug, tyral, this is the trade name of an oral tablet containing l-tyrosine, pyridoxine hydrochloride and niacinamide. The basis for using this combination in allergic diseases rests on "an effort to flood the organism with the natural precursors of epinephrine in an attempt to increase the available stores of this substance."

Dr. Albert H. Rowe says: "We have had the opportunity to use tyral in a large number of patients over a period of many months. No definite evidence of improvement occurred which could not be attributed to the variations of the allergic reactivity itself. It was tried in patients with bronchial asthma, nasal allergy, eczema, recurrent headaches, and other less common allergic manifestations. We have stopped its use because of these very unsatisfactory results."

HARRY L. ROGERS

• PANNICULITIS OR ERYTHEMA NODOSUM

Question: I have a patient, a 32 year old man, with a history of rheumatic fever and a complaint of tender lumps, about the size of an almond, in the subcutaneous tissue of his left leg. Sedimentation rate is 34 mm. in one hour. Physical examination, urinalysis and hematologic study are otherwise negative. Biopsy was reported as typical of Weber-Christian disease or chronic, nodular nonsuppurative relapsing panniculitis. Treatment is the problem. Will cortisone help?

M.D.—British Columbia

ANSWER: We have not used cortisone or ACTH in Weber-Christian disease here, but would not recommend its use. On the other hand, if the patient has erythema nodosum rather than chronic, nodular nonsuppurative relapsing panniculitis, cortisone might be indicated. Histopathologic distinction between the two conditions is not always clearcut.

Surgical Considerations of Certain Hip Lesions

FRANK N. POTTS*

University of Buffalo School of Medicine, Buffalo, New York

Advances in orthopedic surgery have made it possible to deal successfully with many disturbing hip lesions. The tolerance of the body to the inert metals has made many of these advances possible.

PERSONS with hip lesions present themselves for any or all of three reasons: (1) They have pain in the hip; (2) they have instability, or (3) they have varying degrees of stiffness. The cause of these complaints results in lameness, sometimes to the extent that the person cannot work. It is the purpose of this article not to discuss all the lesions that involve the hip joint but rather to consider some more common lesions which merit surgical consideration, to discuss what can be done and to acquaint you with the little-suspected fact that the orthopedic surgeon is aware of his failures. A better understanding of these conditions and their importance to the practitioner, to the patient, to the surgeon, and so often back to the practitioner, should be helpful.

Any disease that involves the bony or the cartilaginous or soft-tissue elements of the hip joint may result in adhesions in and around the joint, destruction of the cartilages, deformity, and therefore an incongruity of the joint. (I will use the word "incongruity" frequently. In this instance it means a want of fitness or harmony, and I know of no better word to describe the mechanical and architectural changes than to say that the parts do not fit.) This incongruity in turn causes pain, limited motion and possibly instability, in direct proportion to the degree of destruction.

*Professor and Head of the Division of Orthopedic Surgery, University of Buffalo School of Medicine, Buffalo, New York.

Presented before the thirty-seventh annual Assembly of the Interstate Postgraduate Medical Association at Cleveland.

Any trauma can result in incongruity of the hip joint.

Any disturbance of the blood supply to the hip joint may result in necrosis and collapse of the subchondral bone, with incongruity, pain, restricted motion and deformity. To a great extent these conditions are not preventable or controllable, and therefore they must be anticipated.

Surgical Lesions in Children

There are few hip lesions in children that call for major surgical intervention. I will not discuss congenital deformities here.

Traumatic separation of the upper femoral epiphysis is readily diagnosed. It can occur in a child of any age. The history of injury, sudden inability to walk, restricted motion, pain (possibly referred to the knee joint) and the roentgenologic examination are sufficient to establish a diagnosis. This condition resembles a subcapital fracture in the adult. It requires manipulation, reduction and in many instances preferably internal fixation done under roentgenographic control. Plaster fixation will give good results, but internal fixation is more certain and the convalescence more comfortable. I have encountered cases in which the separations recur in plaster.

The slower process of slipping femoral

epiphysis occurs in the adolescent. There may be little pain, and this commonly is referred to the knee. There is restriction chiefly of internal rotation. The leg usually is held in a position of external rotation. The slipping process may have progressed nearly to complete separation before the physician sees the child, who usually is of the endocrine obese type. The diagnosis is readily made with anteroposterior and lateral roentgenograms.

This type of slipping cannot be reduced by manipulation. As the process has been slow, it has built up repair at the base of the neck and posteriorly which prevents correction. If the child is seen in the early stage of minimal slipping, internal fixation will prevent further slipping and will hasten the fusion of the epiphysial plate. Technically this is hard to do because of the toughness of the growing bone. Because it is so tough, driving a three flanged nail necessitates so much trauma to the joint that traumatic arthritis may follow. Choice is given to a lag screw which is put in without violence and which provides excellent fixation.

This condition may be bilateral. If the deformity is too great, an osteotomy may be advisable.

Traumatic Dislocation

With the frequency of automobile accidents, traumatic dislocation of the hip has become more common. It is spoken of as a dashboard dislocation. This should be reduced at once and reduction maintained with traction or plaster for about three weeks. We are then obliged to wait two or three years to see if avascular necrosis is going to develop. It may develop much sooner, but it may be late. Avascular necrosis is manifested by an ache in what has been a good, useful hip following the reduced dislocation. The ache increases in severity, restricted motion develops, and the roentgenogram shows mottling and flattening and density changes on the femoral head, with incongruity of the joint.

Should such a patient have been kept off his feet longer? Would such treatment have prevented this complication? Are we justified in keeping a patient from weight-bearing for two years? If we knew for certain that avascular necrosis would develop, the answer might be "yes." This is a serious lesion which to date is not amenable to repair or prevention. I have a feeling that the damage is done at the time of the dislocation, that the blood vessels are damaged, and that resting the part and protecting it for a long period of time does not give any assurance of an earlier revascularization. It is my opinion that we have to wait and see in such cases.

The severe damage to the blood supply of the hip at the moment of dislocation may so deprive the head of its nourishment that it will slowly die.

It is amazing how well some of these persons walk in spite of extreme radiographic changes. The lesion must be treated on the basis of symptoms alone.

Dislocations Complicated by Fractures of the Acetabulum

It may be impossible to reduce and maintain dislocations of the hip complicated by fractures of the acetabulum, as the fragments may be so large and so placed as to eliminate the element of support. These hips must be opened and reduced and the fragments fixed so as to restore the shape of the acetabulum. Too many of these have been accepted as they were, with inadequate restoration of the acetabulum and with the resultant painful, unstable and restricted motion in the hip, prone to redislocation or luxation.

A better understanding of the posterior approach to the hip joint with the excellent exposure it affords makes this type of surgery fairly easy, with much better chances for resultant good function.

Osteoarthritis

The slow, insidious progress of osteoarthritis eventually brings the victim to his physician. His appeal is for relief of pain, for an explanation of the weakness about the hip, and for a solution to the problem of increasing stiffness, cumbersomeness and the inability to do things he has done for years. He does not realize what a marked restriction of motion he has. Flexion or adduction deformities may be developing, and these may be

more marked on one side than on the other. Good roentgenograms demonstrated to him will explain but they will not relieve; the explanation that he has had this condition for years, and that the process will become worse, shocks but does not help him. His earning capacity is involved and his future hazardous. He asks for help. We tell him we cannot control the disease but that plans can be offered with reasonable assumption of some benefit.

Many osteoarthritic hips that are painful have surprisingly good function. If we could ease this pain, the patient could go on for an indefinite time. Efforts to do this have been made for years, with varying degrees of success. Injections have offered only temporary satisfaction. If the hip joint could be denervated the pain should be relieved. This procedure has been carried out with enthusiasm, but this enthusiasm wanes as time goes on and the good effects are shown not to be permanent.

The nerve supply to the hip joint is very extensive, both anteriorly and posteriorly. It remains a question whether this pain comes from the diseased articular surfaces or from the capsule, or from both. It is not easy and probably not possible to find all the nerves supplying the hip joint. However, the procedure is not a disabling one and there is some chance that sufficient relief will be obtained to justify the operation. It is especially valuable when both hips are painful and when the patient is elderly and a poor candidate for reconstructive surgery. Its failure does not preclude other types of surgery.

Value of Arthrodesis

Certainly a stiff hip is a pain-free hip. Consideration of arthrodesis of the hip may be given if only one hip is involved, if there is minimal evidence of osteoarthritis elsewhere, if the spine is not too extensively involved and is movable to compensate for the stiff hip with some lumbar motion, and if the patient is fully aware of the inconvenience caused by a stiff hip.

It is difficult to fuse a hip and it necessitates a long period in bed and in plaster spica. The age of the patient, his temperament and his general physical condition all must be evaluated before the procedure is done.

It was thought at one time that much of this could be avoided by the Watson-Jones method of driving an 8 inch, three flanged nail up through the shaft, the neck and the head and into the thick part of the ilium above the acetabulum. This procedure is not difficult nor does it necessitate such long periods of hospitalization, but unfortunately it has not been as satisfactory as had been anticipated. It has the advantage, as has denervation, that if it fails there are other procedures that can be done.

Arthroplasty

If these fairly conservative surgical methods have failed, more radical plans may be suggested. The mould or cup arthroplasty designed to substitute metal surfaces for the irregular head and acetabulum will allow weight-bearing and good motion without pain. It is a very good procedure with a wide application of usefulness in hip lesions. It sacrifices little bone and calls for active cooperation by the patient in restoring motion and muscle power. It was designed originally for the stiff hip from any cause other than tuberculosis. Motion is sometimes reduced postoperatively, and pain may return. However, if a reasonable range of motion—say, 40 degrees—and a pain-free, stable hip results, it certainly has value.

Prosthetic Artificial Head

Similar in some ways to arthroplasty is the prosthetic or artificial head. There are many mechanical differences. A great interest has been shown in this plan since the Judets reported on their series of over 500 cases. Their thesis was based on supplying a substitution for the diseased or deformed head in an acetabulum, and in so doing relieving pain, allowing motion and affording stability. The method has wider application in that it can be used in cases in which there is considerable loss of neck.

The literature on this subject goes back several years, but the plan was never popularized until the Judets presented their large

series. Some of their patients had been under observation for five years. Their plan was to remove most of the rough, hard, infiltrated capsule, remove the part of the head within the acetabulum, remodel the acetabulum to fit the prosthesis, and then fasten the prosthesis to the prepared portion of the neck. Early weight-bearing and motion are started when the soft tissues are healed, and use is demanded in about 12 days.

The probabilities are that the Judets with their large number of cases are more conservative in their selection than those who follow and try to improve. Following the lead provided by the Judets, others have widened the scope of usefulness and have by necessity designed new types of prostheses. Besides the original acrylic, later prostheses were made of NYLON®, stainless steel and VITALLIUM®. Much might be said in favor of the metal heads. Hip reconstruction by means of prostheses is new, and only time will tell if the bone will tolerate this foreign body indefinitely.

A possible disadvantage of the procedure is that it is likely to be final. Reconstruction after the loss of the head and neck certainly would be a serious and difficult problem. When the stock is used up there is nothing to work with. It must be said, however, that increasingly large numbers of favorable reports are appearing.

Fractures of the Hip

Operative treatment of hip fractures is an accepted procedure. It is far superior to the older conservative treatment of plaster or traction. Only a few elderly patients do not stand this type of surgery well. Proper preparation and aftercare make this a comparatively shockproof type of surgery. Reduction of fragments and internal fixation make convalescence more comfortable. Nursing care is greatly simplified, and the possibility of functional result greatly increased. The farther the fracture is away from the head, the better the opportunity for union.

There is no panacea for fractured hip. Many well reduced and adequately fixed fractures do not unite. Many unite but later develop avascular necrosis of the head, with pain, limp and stiffness. It must be said, however, that there is a better chance of a good result with good reduction and fixation.

We should not be blind to the failures, and these should be discussed freely with the family. The prosthetic head may be the answer in some cases. Certain of the fractures, such as the subcapital ones, might well be treated by means of the substituted head at once. This might be the method of choice in aged persons with short life expectancies and with known hazardous fractures—one that we fear will not unite, such as a subcapital fracture.

All the plans mentioned are well designed. All fail at times, but fortunately all do not fail all the time. We are dealing with a serious pathologic condition that justifies serious consideration and good surgery. The outlook for painful hip, of whatever cause, improves.

A comminuted intratrochanteric fracture will unite without open reduction and fixation. However, a long period of traction is necessary, and if the patient can be made more comfortable, open reduction and fixation are, of course, commendable. It has often been my experience in such cases that there are unrecognized fracture lines; when the hip is opened we find that just where we would like to put a nail or screw there is another fracture line prohibiting it. Our policy always has been to be prepared to back out and use skeletal traction. That is as much a part of our setup in the preparation for fracture treatment as the material for internal fixation.

Operation for Coronary Artery Disease

CLAUDE S. BECK*

Western Reserve University School of Medicine, Cleveland

The primary problem of coronary artery disease concerns red blood to the heart muscle. Operative procedures can accomplish this objective. Two operations have been developed for this purpose. One consists of making intercoronary channels by producing an inflammatory reaction on the surface of the heart. Ligation of the coronary sinus is also carried out in this operation. The second operation consists of placing a vein graft between aorta and coronary sinus. Both of these operations are beneficial. One hundred operations have been done. The clinical results have been excellent. Mortality can be kept around 10 per cent depending on the type of patients selected for operation.

CLAUDE S. BECK

WITHIN the past several hours I had an experience which demonstrates the changing picture in medicine today. Last evening before I left the hospital I saw a patient, a 62 year old woman, who had mitral stenosis. She had had four embolic phenomena. This morning at 10:00 o'clock she had another cerebral embolus, and at 1:00 o'clock we did an emergency operation on her heart, amputated the auricular appendage which was about 3 inches long and was the source of her emboli, broke open her mitral valve and restored the circulation in her heart to practically normal. Yesterday such an emergency operation was revolutionary in medical thinking; today it becomes accomplishment.

I am going to describe two operations for coronary artery disease which may be in a similar category—not quite established today, perhaps, but to be established most certainly tomorrow. In my opinion these operations are established now. The first consists of producing inflammation on the surface of the heart by abrading the surface, using a chemical irritant such as asbestos powder. This opens up the intercoronary channels, crossroads between one coronary artery and another, so that if one artery becomes blocked off there is some circulation which can get into the dry area of the heart. This operation also involves putting a ligature on the coronary sinus and elevating the venous pressure in the heart. This is beneficial to the circulation also, because it produces congestion and makes more oxygen available for the potential dry areas.

The second operation I wish to describe consists of laying a graft between the aorta and the coronary sinus. We take a piece of vein graft from the patient's arm, open the aorta, sew the graft to the aorta and to the sinus, and create a fistula so that blood runs from the aorta through the graft into the coronary sinus, and most of it, in the dog anyway, runs out into the auricle. Three weeks later we put a ligature on the sinus where the blood leaves the heart into the auricle so that it doesn't all run out of the fistula; some of it remains within the venous system of the

*Professor of Cardiovascular Surgery, Western Reserve University School of Medicine, Cleveland, Ohio.

Presented before the thirty-seventh annual Assembly of the Interstate Postgraduate Medical Association at Cleveland.

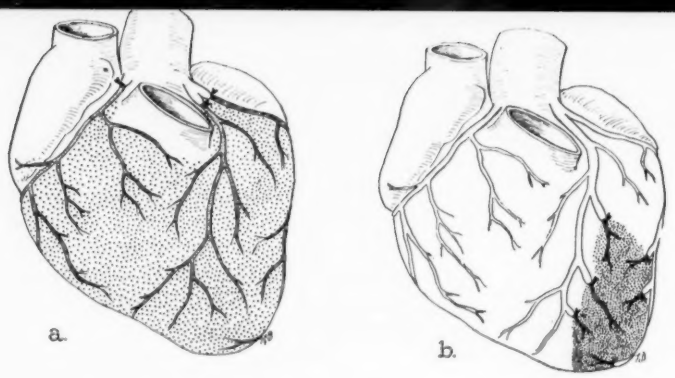

FIGURE 1. Two types of coronary artery occlusion. A. Partial occlusion of three major arteries. This may be tolerated by the heart. B. Complete occlusion of six arteries to a small area of the left ventricle. This produces a trigger which in turn produces ventricular fibrillation and death. A blood bath to this area is a prophylactic measure.

myocardium and in that way benefits the heart.

This operation is beneficial in three ways: (1) It actually produces retrograde flow from the aorta through the graft, through the venous system in the heart and through the capillary bed where it gives up its oxygen; (2) intercoronary channels are created in the heart, communications from one coronary artery to another, and (3) blood is delivered in that extremely rich venous network on the venous side of the capillary bed, and oxygen can diffuse from these small veins and become available to the myocardium without actually going through the capillary bed.

The physiologic aspects of these operations are important, because knowledge of them gives us confidence in evaluating the results on patients. We have to do new thinking on the coronary problem; we have to think in terms of a dry area in the heart, an ischemic area; we have to think in terms of intercoronary channels; we have to recognize that 4 or 5 cc. of red blood into an area of the heart muscle is extremely beneficial and can keep the heart beating.

If four or five ligatures are placed on the small superficial vessels to an area of the myocardium the size of a quarter, that heart will go into fibrillation, a dry area is created and as a result the heartbeat is destroyed. You can cut down the circulation in the heart to a considerable extent by partially occluding the three major vessels and that heart will continue to beat, but the localized ischemia is a very unfavorable situation (Figure 1). When dealing with coronary artery disease, we can say that if that same amount of blood can be evenly distributed over the heart, the patient is better off than if that same amount of blood through the diseased arteries were unevenly distributed throughout the heart muscle where there are peaks in ischemia. Those peaks in ischemia produce many fatal heart attacks.

The nature of the coronary vessels in the dog is very important to the maintenance of the heartbeat. They do not overlap, and the various coronary arteries are not joined together by intercoronary channels. If the surgeon could produce these in the human heart, he could protect that heart to a great extent. These intercoronary channels permit blood to be distributed evenly throughout the heart. I cannot overemphasize the importance of that even distribution.

Coronary arteries are essentially end arteries. If one coronary artery is injected with a mixture of barium sulfate and gelatin, practically none of the mixture passes beyond the arterial bed of the injected artery (Figure 2). If the descending ramus of the left coronary artery is ligated during life and then transected distal to the ligature it will be found that only a small amount of blood emerges from the artery in a retrograde position. This small quantity of blood comes from intercoronary communications, and because of this anatomic arrangement the heart is predisposed to the development of a trigger or an infarct when a major artery is occluded. Conversely, if some intercoronary communications are present, then ligation of one of these arteries can be done without producing a trigger or a necrosis of muscle.

Several years ago my associates and I carried out a study in which we attempted to increase vascularity and distribute blood to every part of the heart by producing inflammation on the heart surface. We exposed the heart by operation and abraded or scraped off the epicardium, and closed the incision. One to three weeks later we again exposed the heart and ligated the descending ramus

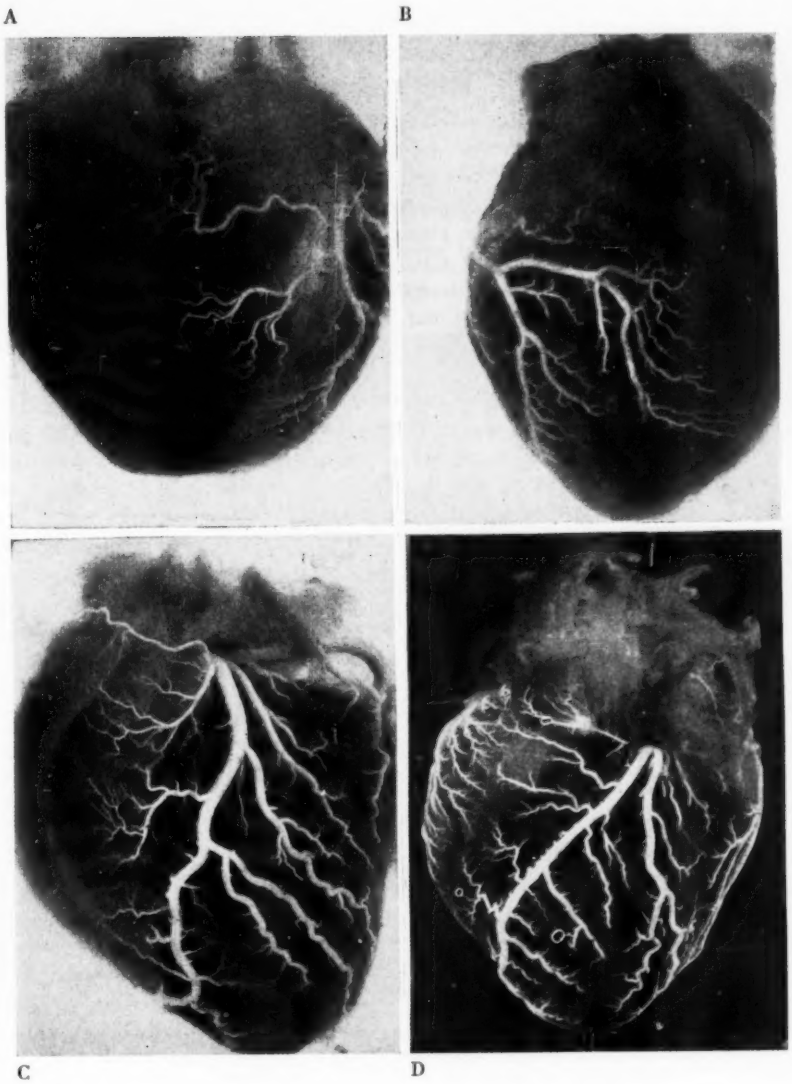

FIGURE 2. Normal hearts; coronary arteries injected with mixture of barium sulfate and gelatin. The coronary arteries are practically end arteries, but a few intercommunications can be found in some specimens. A. Left circumflex artery injected. Small amount of barium sulfate can be seen in descending ramus of left coronary artery. B. Circumflex branch of left coronary artery injected. C. Common left coronary artery injected. D. Three coronary arteries injected.

(Figures 1, 2 and 3 from Beck, C. S.: Principles underlying the operative approach to the treatment of myocardial ischemia. Ann. Surg. 118:788-806 [November] 1943. Reproduced courtesy of J. B. Lippincott Company, publishers.)

of the left coronary artery at its origin. This experiment was performed on 50 dogs, and ligation of the artery alone was done in another group of 50 dogs. The mortality rate in the control group was 70 per cent; that in the group in which abrasion and ligation were done was 38 per cent. Intercoronary anastomoses could be demonstrated in many of these abraded hearts (Figure 3), and in some the infarct was

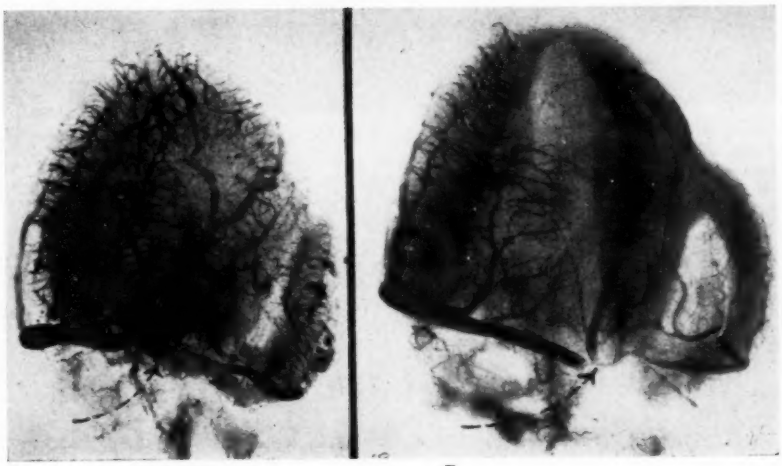

FIGURE 3. Circumflex branch of left coronary artery and the right coronary artery were injected with a mixture of barium sulfate and gelatin. The descending ramus of the left coronary artery is indicated by the arrow. Note the difference in the injection in these specimens. A. A normal heart. B. A heart the surface of which was abraded at operation two weeks before killing the animal for injection. The degree of injection in the descending artery is an indication of the intercoronary communications present in these hearts. (A) shows the greatest injection out of a group of 10 normal hearts, and (B) shows the greatest injection out of a group of 10 abraded hearts.

small or there was no infarct present at all.

We also attempted to produce extracoronary communications by grafting tissues on the heart, the basis for this experiment being to get blood from the outside of the heart to go into the coronary circulation. Specimens obtained in this experiment demonstrate anatomic communications, but I don't know how important these specimens are. They do not tell us how much blood goes across between the heart and the graft, or which way it goes. Also, you cannot always count on these extracoronary communications developing, and very often they do not.

Let's turn to experiments involving tying off the coronary sinus and also putting red blood in the coronary sinus. In a study made by Dr. Eckstein, the circumflex artery was tied off in 20 normal dogs, and the mortality rate was 70 per cent within an hour. In a group of dogs in which red blood was put in the coronary sinus and the same test artery was tied off, there were no deaths within an hour. An A-V fistula made elsewhere in the circulation has no effect on the mortality after the test artery is tied off. So it is not the fistula that reduces the mortality from 70 per cent to zero; it is the red blood in the sinus. Elevating the sinus pressure and tying off the test artery result in a mortality rate of 30 to 40 per cent. These studies are interesting in that the red blood gives tremendous protection against ventricular fibrillation—zero compared with 70 per cent—and even elevation of the pressure in the coronary sinus provides protection by engorging the heart with blood and making oxygen more available to the heart muscle. I don't see why medical men or surgeons or physiologists should have difficulty in accepting or interpreting these experiments, and I don't think we can go along year after year without paying attention to this work.

Let's consider the question of putting a free graft of vein between the aorta and the coronary sinus to put the blood right into the venous system of the heart (Figure 4). The destruction produced by tying off a test artery in the normal heart is seen in the three specimens in Figure 5, which shows the hearts of the 3 dogs which lived following ligation. A large infarct is present in each specimen. An entirely different picture results when you place a graft between the aorta and the cor-

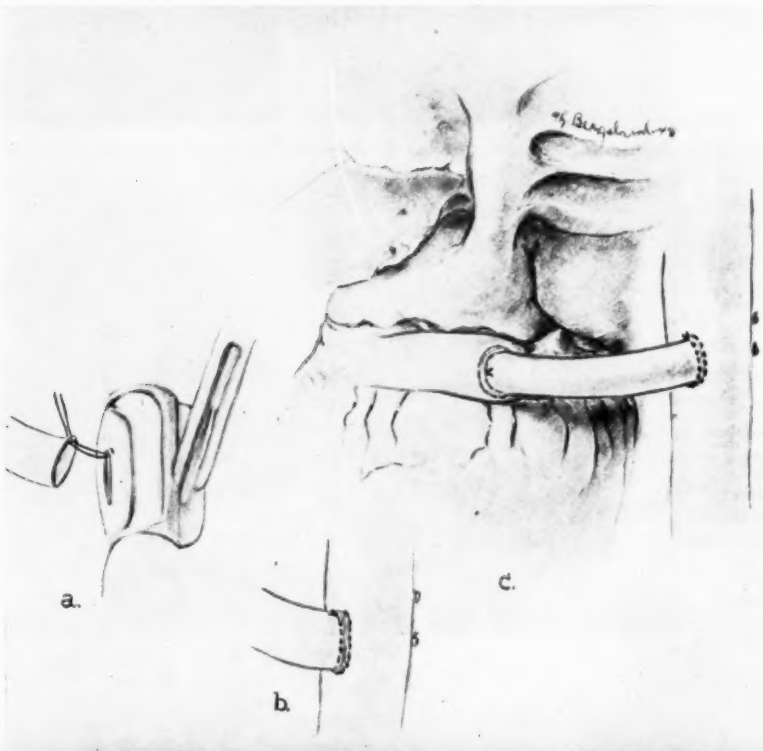

FIGURE 4. Free graft of vein between aorta and coronary sinus of the dog. The aorta is dissected free for a distance of about 3 cm. Two intercostal arteries are cut. The special clamp is placed on aorta as in (a), allowing some blood to pass the clamp. The aorta is opened by incision about 5 mm. in length. An everting mattress suture is placed between graft and aorta at each end of this incision. Anastomosis is completed by a continuous everting suture as in (b). (c) shows the complete operation.

onary sinus (Figure 6) and then tie off the same test artery. I repeat, I don't see why there should be any difficulty in interpreting this or in accepting it as proof that this operation is beneficial to the heart.

We have ligated the descending coronary artery in a group of 37 dogs which had a graft between the aorta and the coronary sinus, and only 4 dogs died. In normal dogs the mortality rate for ligation of this artery is 70 per cent, and the infarcts are large in the dogs that recover.

Eckstein and Leighninger have carried on experimental work with these grafts. Leighninger has spent three years in my laboratory working on this project. It has been shown that when a circumflex artery is tied proximally and cut distally in a normal dog, the amount of retrograde flow is 2.8 cc. per minute. When we do the same thing after putting red blood into the sinus, the amount of flow is 7.6 cc. per minute. This 7.6 cc. is venous blood; the 2.8 cc. is red blood. The 2.8 cc. is intercoronary blood and the 7.6 cc. is blood which comes from the aorta; it is blue; it has given up its oxygen.

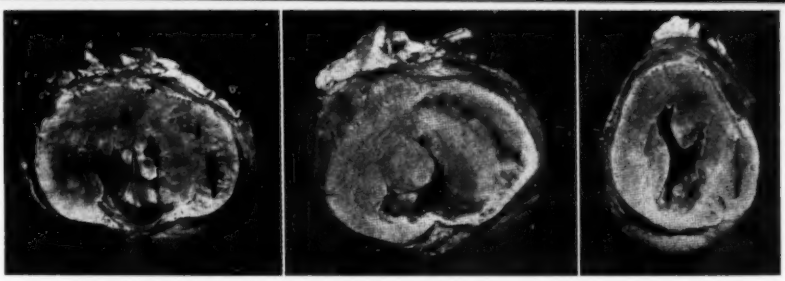

FIGURE 5. Infarcts in the control group. The descending ramus of the left coronary artery was ligated in a group of 10 dogs. Only 3 animals survived this ligation and each of these specimens showed a large infarct. Compare with Figure 6.

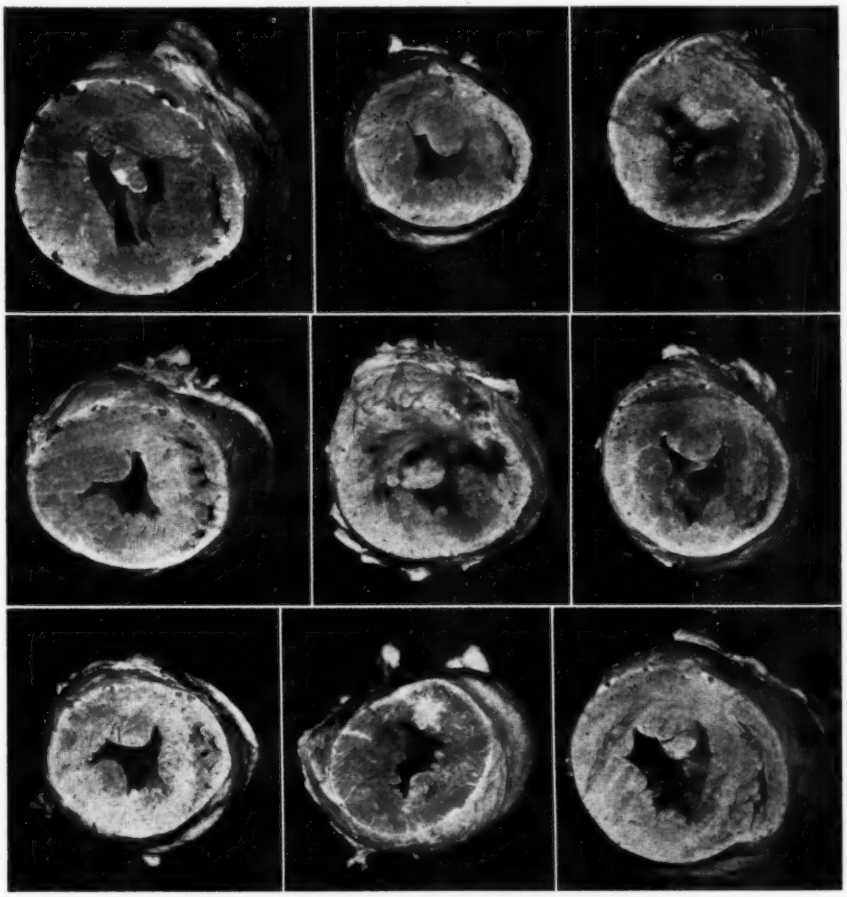

FIGURE 6. Infarcts in group with anastomosis between artery and coronary sinus. The descending ramus of the left coronary artery was ligated in a group of 10 dogs. There were no immediate deaths after this ligation. In 4 specimens there was no infarct. In the other specimens destruction was minimal and appeared in scattered areas. In no specimen was extensive destruction of myocardium found as occurred in each of the control group. Each specimen in Figure 5 and Figure 6 is mounted in the same relative position. The infarct is inferior and between left and right ventricles. The grafted artery can be seen in several specimens. In the middle specimen of the bottom row a scar is seen anteriorly. This occurred because the circumflex ramus of the left coronary artery was accidentally ligated at the time of anastomosis. The small infarct following ligation of the descending ramus of the left coronary artery lies opposite this scar in a papillary muscle.

(Figures 4, 5 and 6 from Beck, C. S.: Revascularization of the heart. Ann. Surg. 128:854-864 [October] 1948. Reproduced courtesy of J. B. Lippincott Company, publishers.)

To have done so, it must have gone through a capillary bed, and therefore, this is the first proof that blood from the aorta can be put into the venous system of the heart and be proved to go through the capillary bed and out the severed artery. There can be no question about interpretation of that experiment. Other experiments have produced retrograde flows of 20 to 30 cc. per minute; 20 cc. from any coronary artery is very protective; 5 cc. is protective; 10 cc. is very protective and will keep a heart from going into fibrillation.

This retrograde flow from aorta through capillary bed and out the severed artery begins to subside after four to six weeks, and the intercoronary flow begins to increase. At the end of a year, there still is significant protection provided by intercoronary flow.

Who among us understands the importance of 5 cc. of blood to, say, half of the left ventricle per minute? Well, 7.6 cc. minus 2.8 gave Eckstein a reduction in mortality rate from 70 per cent to zero. That is how important it is.

The operation of placing a graft between the aorta and the coronary sinus used to be very difficult, but it now can be done in two and a half hours and I think any good surgeon can train himself to do it. The patency rate of these grafts in the dog has been 96 per cent, and one of the men who spent two years in experimental study of the procedure had a series of 104 successive operations without a death. Our laboratory is devoted solely to the development of this operation. The work is supported by the Public Health Service and the Cleveland Heart Society. The coronary problem to us is cubic centimeters of red blood to the heart muscle. So little has been written about this subject, namely, blood to the heart muscle, and so few persons have done any work on the problem that one could reasonably wonder just what is the problem in coronary artery disease. *The problem is blood to the heart muscle.* Let us not forget that. If we are going to do anything for the patient with coronary heart disease, we will have to give the heart a blood supply.

We are over the initial experimental period with this procedure, and we have gone far enough with the clinical results to know that the operation is beneficial to patients.

The vein graft becomes thrombosed in some cases. When this occurs we have to do the alternative operation, which consists of ligating the sinus and producing inflammation on the surface of the heart. In order to overcome thrombosis, we are going to try to attach the coronary sinus directly to the aorta. We have been making instruments for use in doing this. We haven't done it yet, but in many cases the aorta and the coronary sinus touch, and we are going to cut a little round ring out of the aorta and try to bring the two structures directly together. Perhaps that will overcome the thrombosis problem.*

Coronary artery disease kills twice as many people as does cancer. It is not just an old man's disease. It strikes in the fifties and in the forties when a man is at his best. It will kill 1 of every 4 men in this room. The amount of money spent on cancer research is many times greater than that spent on coronary artery research.

The mortality rate for this procedure will depend, I think, on the selection of the patients. We lost 8 of the first 12 patients. In the next 59 cases there were 12 deaths, or about 20 per cent. None of the last 10 patients has died, and only 1 death has occurred in the last 17 cases. I think every patient should be digitalized before operation, because it reduces the tendency to fibrillation. I think also that the mortality rate for this operation can be reduced to about 10 per cent or maybe even less. Recently we sent letters to 17 patients who had open grafts. Replies indicated that more than 70 per cent are able to work; 88 per cent have less pain.

Indications for operation include positive diagnosis, one or more arterial occlusions, pain, and some physical incapacity; also, we like to have patients who have had the disease for some time. Contraindications are marked enlargement of the heart, failure, dyspnea at rest, progressive disease in a young man, a recent infarct, and hypertension; the age limit is about 62 years.

*Since this address was made, this has been accomplished and the patency rate has been improved.

Kaposi's Sarcoma (Angioreticulomatosis)

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Kaposi's sarcoma is a disease which, while not uncommon, is frequently not recognized. An attempt has been made to present the various features of this condition which are of value in establishing its diagnosis. Sixteen cases which have been under our observation are reviewed.

FRANCESCO RONCHESE

ARTHUR B. KERN

MANY names have been given to this disease since its original description by Kaposi[1] in 1872 (Table 1). A primary reason for multiplicity and variety of titles of a not uncommon disease is the uncertainty of its etiology. We are inclined to consider Kaposi's sarcoma a reticuloendotheliosis and would prefer a title like "angioreticulomatosis." In principle we believe that the adding of personal names to syndromes and diseases is a plague affecting medical nomenclature. Only exceptional names or titles, we believe, should be retained out of respect to history, brevity, clarity and quick general understanding of what the reader may expect to find discussed in the text. Kaposi's sarcoma is a title covering these requirements. Lengthy subtitles can be added between parentheses ad libitum.

We would like specifically to point out the fact that use of the name "Kaposi's disease" should be abandoned, since this title has generally been applied to an entirely different disorder, xeroderma pigmentosum.

It has been repeatedly stated that the condition occurs predominantly in Jews. McCarthy and Pack[2] recently reported that of 36 cases of Kaposi's sarcoma, 30 patients (83 per cent) were Jews or Italians. Beerman,[3] in his monograph on tumors of the skin, pointed out that this disease occurs chiefly in Italians and Jews. However, Azulay,[4] in an excellent review of the subject, stated that he had no Jews in his series of patients from Brazil and cited DeAmicis' observation of not a single Jew among 50 patients. Among the large series of 356 cases Dörffel[5] collected from the literature, only 45 patients were Jewish. There were 111 Italians, 50 Russians and 20 Poles; most of the Jewish patients were born in Russia or Poland but are not included in the figures given for those countries. The remainder of the 356 cases represented nearly every nationality in the world. Kocsard[6] reported the only case of Kaposi's sarcoma in an Oriental.

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TABLE 1

SOME OF THE TITLES USED TO DESIGNATE KAPOSI'S SARCOMA

YEAR	AUTHOR	TITLE
1869	Köbner	Multiple cutaneous metastatic sarcomatosis
1872	Kaposi	Idiopathisches multiplex pigmentsarkom der haut
1877	Tanturri	Sarcoma idiopatico parvicellulare teleangettasico pigmentato della pelle
1882	DeAmicis	Dermatopolimelanosarcoma idiopatico
1884	Hardaway	Sarcoma cutis (first paper in the United States)
1894	Kaposi and Köbner	Sarcoma idiopathicum multiplex hemorrhagicum
1894	Unna	Acrosarcoma multiplex cutaneum teleangectodes (Hebra-Kaposi)
1898	Tommasoli	Primitives haemorrhagisches acrosarcoid
1899	Pospelow	Acroangioma haemorrhagicum
1905	Pelagatti	Sarcoma molteplice primitivo di Kaposi
1913	Martinotti	Sarcoma haemangioendoteliale intravascolare
1927	Hamdi and Halil	Perithelioma multiplex nodulosum cavernosum lymphangectoides cutaneum
1930	Radaeli	Angio-endotelioma cutaneo (since his first paper in 1904 Radaeli advanced the idea of an angioendotheliomatosis in contrast to the other dermatologic schools)
1931	Tramontano and Fittipaldi	Emoangioendotelioma del Kaposi
1934	Dupont	Maladie de Kaposi
1941	da Cunha Motta	Hemangio-reticulo-endothelioma
1942	Bessone	Angioendotelioma cutaneo
1949	Azulay	Angioreticulosis Kaposi
1950	Grinspan	Reticulo-endoteliosis angiogena de Kaposi
1950	Nödl	Angiomatosis Kaposi

Prior to December 1949 only 10 instances of the disease in the Negro had been recorded in the literature.[7-16] Since then, however, there has been the report by Syrop and Krantz[17] concerning a Negro with oropharyngeal lesions, and the reports of 6 cases in Negroes of the Belgian Congo.[18-19] In addition, Kaminer and Murray[20] found 33 cases of Kaposi's sarcoma in the Negro recorded in the files of the South African Institute for Medical Research for the 1942-1949 period. The latter authors suggested that anthropologic differences between these two branches of the Negro race might explain the greater incidence in the South African Negro as compared with the American Negro. We wonder whether it might not be due in part to the greater difficulty for the physician in this country to make the clinical diagnosis of Kaposi's sarcoma on a pigmented skin. In support of this concept is the report of Hazen and Freeman[21] who have had considerable experience in the care of cutaneous diseases of the American Negro. In a series of 21,000 Negro patients they observed 129 cases of cutaneous carcinoma of which 3 were Kaposi's sarcoma. It was their opinion that this condition is of equal frequency in Negroes and in white persons.

There were 325 men and 21 women in Dörffel's series,[5] a ratio of approximately 15:1. Choisser and Ramsey[22] in 1939 collected 434 cases of Kaposi's sarcoma from the literature and observed the same sex ratio. All investigators have noted the greatest incidence in the sixth and seventh decades of life. The reviews of Dörffel,[5] Choisser and Ramsey[22] and Kren[23] emphasize the fact that office workers and professional men are less frequently affected than are laborers and out-of-door workers. McCarthy and Pack,[2] however, found that the occupations of patients in their series were too diversified to support the observations of these previous authors, but did note a prevalence of occupations which required the individual to be on his feet for a long time.

A small brown, blue or red macule is generally the first manifestation of the disease. Nodules and plaques subsequently develop and both are present in most fully developed cases; occasionally only one type is present or one may predominate. The nodules are firm and vary from a few millimeters to 2 or 3 cm. in diameter. They may be few in number, as in the cases cited by Symmers[12] (one lesion in 2 and two in the third), or may be quite numerous, Babes[24] having observed 450 in 1 patient. The tumors may be discrete, or several may coalesce to form large masses. They, as well as the plaques, tend to be bluish, bluish red or reddish brown in color. The plaques, which are usually well demarcated, may be small or may reach a size of 20 cm. or more. In the early stages these are only slightly indurated, but as the lesions age the induration increases. Telangiectases may be present in the skin overlying the tumors. Nodules and plaques have been observed developing in areas of purpura. Lesions simulating cysts, bullae[25] and lymphangioma circumscriptum have been reported, as have deeper angiomatous tumors resembling simple angioma and granuloma pyogenicum. In rare instances the disease may appear in the form of tumors from the start (the "d'emblée" variety).[26]

The eruption usually begins on the hands and feet, more frequently the latter. However, it may start anywhere on the body. The disease tends to be unilateral early in its course, later becoming bilateral. The lesions may be few in number and localized to one area—as in the case reported by Gougerot and Burnier,[27] in which they were limited to the palms—or there may be extensive involvement not only of the extremities but also of the face, trunk and the visible mucous membranes.

A common feature of Kaposi's sarcoma is swelling of the extremities. This may be the first evidence of the disease, as in case 1 of Stats'[28] report, in which the patient developed symmetric pitting edema of both lower extremities six months before nodules of Kaposi's sarcoma appeared in the edematous tissues of the legs. In some cases the pitting edema stops abruptly just below the knee or elbow so that the leg and foot or the forearm is markedly enlarged while the thigh and arm are of normal size. In others the entire extremity may be involved. The cause of this type of swelling, which resembles that due to congestive heart failure, is unknown. Enlargement of an extremity in some patients is due to the large, locally infiltrative plaques; the swelling may be increased by the tumors' obstruction of lymphatics. Symmers[12] believes that the elephantiasis is due to thickening of the collagenous tissue in the neighborhood of the skin nodules with a resultant impairment of lymphatic drainage.*

FIGURE 1. The typical appearance and distribution of lesions of Kaposi's sarcoma.

*Cajaffa (Alterazioni ossee nel morbo di "Kaposi," Atti del XXXV Congresso della Societá Italiana di Ortopedia e Traumatologia, October 1951) has added a new sign of Kaposi's sarcoma: osteoporosis. He described 3 cases with bone rarefaction corresponding to the oldest areas of vascular tumoral involvement. Six of our patients and 2 others not included in this group, all advanced cases with considerable edema and numerous vascular tumors, showed no bone pathology. Bone studies could not be done in the other 10 of our series.

FIGURE 2. Plaques and nodules of the extremities and face.

Visceral involvement occurs in about 10 per cent of cases.[29] The internal organs affected, in order of frequency, are the gastrointestinal tract (all portions), liver, lungs and superficial and deep lymph nodes. However, almost every organ may be the site of the disease. In those cases where there is lymph node or splenic enlargement, histologic examination of these tissues reveals tumor nodules in some and nonspecific change in others. It has been suggested that the latter represents either a nonspecific inflammatory reaction or possibly an early stage in the development of Kaposi's sarcoma.

Visceral involvement most commonly follows the appearance of cutaneous tumors. However, in Paólini's case[30] the reverse was observed. A nodule of the gum was noted several months before skin manifestations in the patient described by Pearce and Valker.[31] Barringer and Dean[32] reported 2 cases in which the original lesion was on the glans penis, while Van Cleve and Hellwig[33] and Goldschlag[34,35] observed primary development in the inguinal lymph nodes. There was initial involvement of the pharynx and larynx in the patient described by Bilancioni.[36]

Visceral lesions in the absence of cutaneous manifestations are extremely unusual, and Symmers[12] questioned the existence of such a condition. Greppi and Bettoni[37] reported a case in which the glans penis was the first site, with later involvement of the inguinal nodes; cutaneous lesions were not observed and, at autopsy, tumor was also found in the psoas muscle and lungs. Choisser and Ramsey,[22] Weller,[38] da Cunha Motta[39] and Aegerter and Peale[13] have reported a total of 4 cases in which the disease was limited to the heart. Tedeschi and associates[29] observed a 17 year old boy who died following intestinal intussusception and whose autopsy revealed Kaposi's sarcoma only of intestine, liver, spleen and lymph nodes. The most recent evidence of the fact that this disease can be limited to the viscera was presented by Nesbit, Mark and Zimmerman[40] in their report of a case of disseminated visceral lesions in the

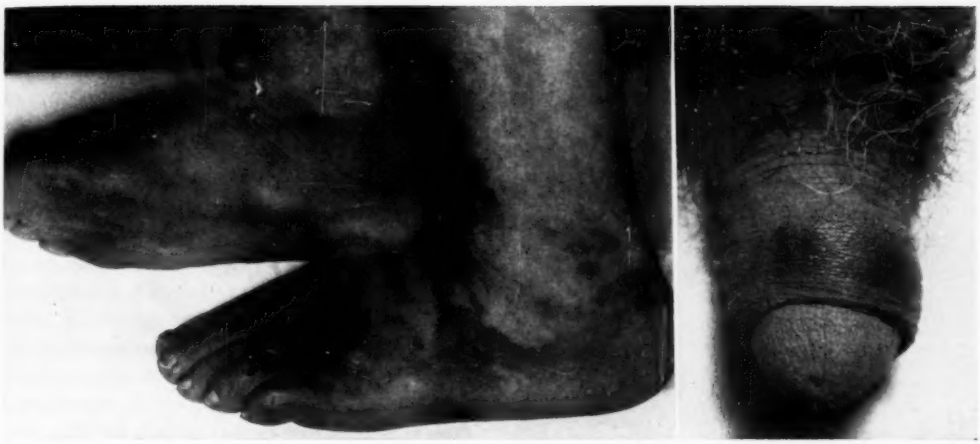

FIGURE 3. Typical clinical appearance of Kaposi's sarcoma on feet. Unusual location is the foreskin. This patient also had nodules on one auricle. Both auricular and penile lesions involuted spontaneously.

absence of cutaneous manifestations.

It was originally believed that visceral tumors were the result of metastasis. However, the observation of patients in whom extracutaneous lesions preceded skin involvement as well as those in which the disease involved only the internal organs suggests that Kaposi's sarcoma develops in multiple primary foci. The fact that the pathologic changes in the viscera histologically mimic the early through late alterations of the skin tumors is additional evidence that new lesions develop not as the result of metastasis but rather through autochthonous origin.

Many authors have commented on the changes of the hemogram which occur in association with Kaposi's sarcoma. Most commonly reported have been secondary anemia and monocytosis, with eosinophilia and lymphocytosis less frequently observed. Flarer[41] noted the presence of an increased number of mononucleated cells which differed from the normal monocytes and lymphocytes of the blood. These he called "abnormal mononucleated cells." He also described them in mycosis fungoides, and their presence in the blood of individuals with that disease was confirmed by Chiale[42] and others. Belloni,[43,44] in discussing the hemograms of his patients with Kaposi's sarcoma, refers to the abnormal mononucleated cells as "Flarer's cells." Bertaccini[45] observed increased reticuloendothelial and monocytic cells in the bone marrow of 2 of 3 patients, each of whom showed monocytosis in the peripheral blood. He called them "abnormal non-classifiable mononucleated cells" practically corresponding to the "abnormal mononucleated cells" of Flarer.

Bessone[46,47] called Flarer's finding a new chapter in the pathology of Kaposi's sarcoma. He confirmed the constant presence, although in varying proportions, of "atypical mononucleated elements," partly of lymphoid and partly of histioid structure. His opinion on their relation to Kaposi's sarcoma is, however, prudently reserved. Flarer's most interesting observations were published in 1936. We have no knowledge of authors other than those cited, and particularly outside of Italy, having confirmed his findings. Nevertheless, Flarer's work plus the occasional observation of the simultaneous occurrence of the disease with mycosis fungoides,[48] Hodgkin's disease[49] and lymphatic leukemia[50-53] have suggested inclusion of Kaposi's sarcoma in the lymphoblastoma group. Businco,[54] because of the two main components, the vascular and the reticuloendothelial, put the disease in the group of the mixed reticuloendotheliomas. He approved of Radaeli's name of hemo-angio-

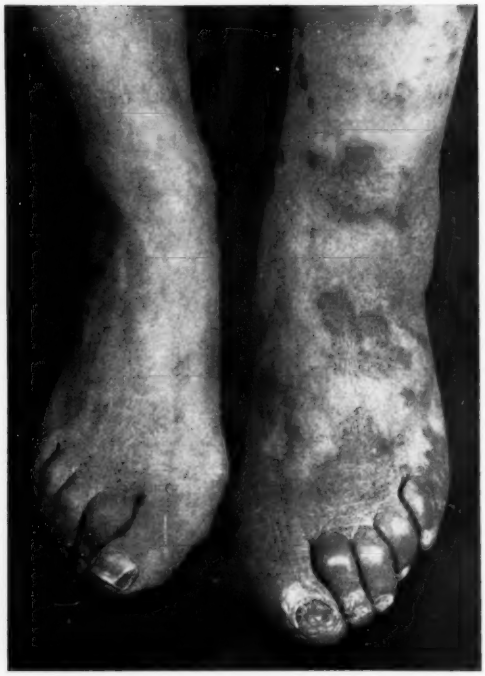

FIGURE 4. Asymmetric eruption, erroneously diagnosed as stasis dermatitis. Note the unilateral edema.

endothelioma and agreed with Flarer, Bertaccini and others in considering Kaposi's sarcoma a reticuloendothelial disease.

The multiple theories as to the nature of Kaposi's sarcoma have been divided into four main groups by Choisser and Ramsey:[22] (1) neoplasm, (2) infectious granuloma, (3) infectious granuloma with neoplastic potentialities and (4) reticuloendothelial hyperplasia. There has been little mention in the American literature of the interesting investigations of Greco and collaborators,[55-57] who studied Kaposi's sarcoma in several members of the same family and concluded that the disease had an infectious, mycotic origin. They called it a myco-hemo-angio-endothelitis with colliquative dermo-epidermic reaction and named the pathogenic fungus *Cryptococcus hoematicon*. European investigators, however, have been unable to confirm Greco's findings. At present it is generally believed that the disease is a neoplasm, some maintaining that it is primary and others that it is secondary to chronic hyperplastic inflammation and granuloma. Among those who believe that the disease is a neoplasm, considerable difference of opinion exists as to the cell of origin. This, to a large extent, is due to the varied histologic pictures which may be observed.

As pointed out by MacKee and Cipollaro,[58] in the early stages the pathologic process tends to be inflammatory, with dilatation of blood vessels and lymphatics, edema, hemorrhage and a perivascular infiltrate of round cells, connective tissue cells and some plasma cells. Later the granulomatous element may predominate, proliferation of small vessels and connective tissue being added. In the late stage the neoplastic features are most marked and, depending on whether the vascular or the connective tissue elements predominate, the picture may be that of an angioma, granuloma, lymphangioma, fibroma, spindle cell sarcoma or angiosarcoma. The pathologic changes which occur in the viscera are the same as those in the skin. Sachs, Azulay and Convit,[59] on the basis of their histopathologic studies, have suggested that glomus tumor, granuloma pyogenicum and Kaposi's sarcoma are all angioblastomas. They further concluded that Kaposi's sarcoma may be subclassified as a systemic angiosarcomatosis.

Diagnosis on the basis of the history and clinical appearance of the cutaneous lesions is relatively easy (Table 2). When the tumors are few in number, involve unusual

TABLE 2

DIAGNOSTIC CLINICAL FEATURES OF KAPOSI'S SARCOMA

1. Most commonly occurs in males
2. Greatest incidence in the sixth and seventh decades of life
3. Predominant involvement of extremities
4. Symmetry
5. Bluish, bluish red or reddish brown nodules and plaques
6. Lesions painless; ulceration uncommon
7. Edema of an extremity common
8. Residual atrophy and pigmentation of lesions spontaneously involuting
9. Slow progress
10. No change, until late, in patient's general condition

sites or do not have the typical appearance, differentiation from various benign and malignant tumors such as angioma, angiosarcoma, granuloma pyogenicum and lymphoblastoma may require histologic studies. The majority of individuals with visceral lesions present no symptoms referable to them and in most cases the lesions are discovered at autopsy. However, hemorrhage, diarrhea, abdominal pain, intussusception and emaciation may be manifestations of the disease. It is in such cases that cutaneous tumors may supply the clue as to the nature of the internal disorder.

X-ray therapy is almost universally accepted as the treatment of choice for Kaposi's sarcoma. Most lesions respond promptly to doses of 75 r, unfiltered, at weekly intervals. Large doses are not recommended. It occasionally has been suggested that an extremity or part of an extremity be amputated to prevent spread of the disease. McCarthy and Pack[2] treated 2 patients with early lesions limited to the penis by amputation of the organ. Two patients in our series, on the other hand, showed spontaneous involution of their penile lesions. If one accepts the concept that Kaposi's sarcoma does not metastasize but that new tumors arise from multiple primary sites, amputation of an extremity or the penis for small, early lesions is about as justified as surgical removal of a syphilitic chancre. Arsenic has been used in the past, and MacKee and Cipollaro[58] recommend its employment during the periods of rest from x-ray. Recently Grinspan[60] reported good results with penicillin in the treatment of 7 patients with Kaposi's sarcoma and pointed out that confirmation of his results would be evidence of the infectious etiology of the disease. Piera[61] administered penicillin to a patient with Kaposi's sarcoma because of a concomitant lung infection; there was initial improvement of the tumor nodules, but this was only temporary. Gougerot and Hewit[62] treated 2 patients with penicillin and observed improvement in 1 and no effect in the other. Witten[63] reported no visible change in 6 patients treated with penicillin.*

*In 2 advanced cases, not included in this series, penicillin was used intensively without any visible effect.

As stated previously, the lesions generally respond to radiation therapy. However, new ones continue to appear, treatment serving primarily to increase the comfort of the patient. Even without treatment, as pointed out by Grinspan,[60] macular, infiltrative and tumoral lesions will occasionally disappear spontaneously, leaving an atrophic scar and pigmentation. Generally the individual lesions do not produce symptoms, but when a great many are grouped on one extremity and there is considerable edema, or when ulceration develops (usually after trauma), there may be discomfort and impairment of function. It was Kaposi's original belief that the disease was rapidly fatal in from two to three years. He later concluded that its course was 5 to 10 years. Dörffel,[5] however, found many cases in the literature in which the patients lived for as long as 25 years. Jacobsen[64] mentioned that the disease may be present even up to 48 years after the onset. In cases of such prolonged duration where death is from intercurrent disease one should speak of death "with" Kaposi's sarcoma and not "from" Kaposi's sarcoma. As summed up by MacKee and Cipollaro,[58] the course of the disease is generally slow but at times it may be rapidly progressive. Their studies indicated that the average duration is from 5 to 10 years, with death generally due to intercurrent disease or some complication but often the result of hemorrhage and emaciation secondary to visceral involvement.

Review of 16 Cases

The records of the Rhode Island Hospital for the past 20 years yielded 15 cases of Kaposi's sarcoma; an additional patient was observed at the Charles V. Chapin Hospital.

Certain general features of each case are presented in Table 3. Fifteen of the 16 patients were men. The average age at onset of the disease was 54.2 years—1 was in the second, 1 in the fourth, 9 in the sixth, 4 in the seventh and 1 in the eighth decade of life. Ten of the patients had been born in Italy, 3 in Russia and 1 each in the United States, England and Lithuania. There were only 4 Jews in the series, 1 born in the United States

FIGURE 5 *(left)*. Unilateral edema and characteristic distribution in patient.

FIGURE 6 *(below)*. Note the unilateral inconspicuous lesions and the conspicuous unilateral edema. This patient has been followed at the Rhode Island Hospital Tumor Clinic during the past 16 years. Recently he was seen elsewhere where the diagnosis of elephantiasis of unknown origin was made. Treatment consisted of an extensive resection of connective tissue and plastic repair. In view of the nature of the disease and the likelihood of recurrence, the benefit of such therapy will be short-lived. This case emphasizes the importance of making the correct diagnosis in patients with edema of an extremity.

and 3 of Russian origin. In those cases where the occupation was known, a decided predominance of laborers and out-of-door workers was evident.

The slow progression of the disease is also apparent from Table 3. Since most of the patients are still living, the figures in the table do not represent the true duration of the disease. Nine of the subjects were treated with x-ray, in 1 case combined with surgery; 1 was treated with radium and surgery, and 1 with surgery alone; 5 received no therapy. Satisfactory regression of tumors with x-ray was observed. In 1 (Case 2) chronic radiodermatitis of the foot and ankle developed as the result of excessive radiation, and amputation was required. Surgical excision of a tumor-bearing area in 1 case was followed by recurrence in the graft, and in another there was recurrence in the scar. Many of the patients reported spontaneous involution of tumors. We wonder whether such involution may not occur as the result of a process which is similar to that responsible for the spontaneous involution of infantile hemangiomas, another vascular disorder.

There were 6 deaths in the series. One patient died at home from unknown causes. For the other 5, the causes were recorded respectively as cerebrovascular accident, hypostatic pneumonia, Kaposi's sarcoma and senile psychosis, cirrhosis of the liver, and carci-

TABLE 3

GENERAL FEATURES OF 16 CASES OF KAPOSI'S SARCOMA

CASE	SEX	AGE AT ONSET	BIRTHPLACE	OCCUPATION	KNOWN DURATION OF DISEASE, YEARS	TREATMENT	COURSE
1	M	75	Italy	Unknown	25	X-ray	Died; cause unknown
2	M	51	Italy	Laborer	12	X-ray	New tumors continue to appear as treated ones involute
3	M	32	Italy	Gardener	15	X-ray	
4	M	63	Russia	Factory worker	10	X-ray	
5	M	60	Italy	Gardener	4	X-ray	
6	M	66	Italy	Peddler	8	Excision and x-ray	Died; hypostatic pneumonia
7	M	57	U.S.A.	Unknown	13	X-ray	Died; cerebrovascular accident
8	F	59	England	Unknown	7	Excision	No recurrence after five months
9	M	56	Italy	Cobbler	Unknown	None	Died; cirrhosis of liver
10	M	57	Lithuania	Storekeeper	8	X-ray	Died; carcinoma of rectum (?)
11	M	53	Italy	Factory worker	2	None	Unknown
12	M	51	Russia	Unknown	8	None	Unknown
13	M	55	Russia	Storekeeper	8	None	New tumors periodically appear
14	M	64	Italy	Laborer	8	None	Died after development of senile psychosis
15	M	51	Italy	Laborer	18	Radium and excision	Periodic development of new tumors
16	M	54	Italy	Factory worker	7	X-ray	Unknown

noma of the rectum. No autopsies were performed. As regards the latter 3 cases, the question arises whether the involved viscera may have been the sites of Kaposi's sarcoma. Nesbitt, Mark and Zimmerman[40] have reported the only case in which Kaposi's sarcoma developed in the brain. Whereas it is most probable that the senile psychosis was due to the usual degenerative changes, it is possible that the mental abnormality was the result of Kaposi's sarcoma arising within the brain. It is also possible that the hepatic changes in the individual with cirrhosis was due to the presence of tumor. However, the history of chronic alcoholism suggests this as a more probable cause.

The patient in Case 10 was first seen in 1931, at which time he showed nodules on the extremities which were clinically and histologically typical of Kaposi's sarcoma. In 1940 he was hospitalized because of diarrhea and rectal bleeding. Barium enema, gastrointestinal series and rectal and proctoscopic examinations failed to reveal the source of the bleeding. Five years later he was again hospitalized because of rectal bleeding. Examination at this time revealed an annular ring near the anal sphincter, and the diagnosis of carcinoma of the rectum was made. Death occurred three days after admission. Although carcinoma cannot be excluded as a possible cause of death, gastrointestinal symptoms extending over a five year period in an individual with Kaposi's sarcoma suggests that the tumor of the bowel may have been another manifestation of this disease. Unfortu-

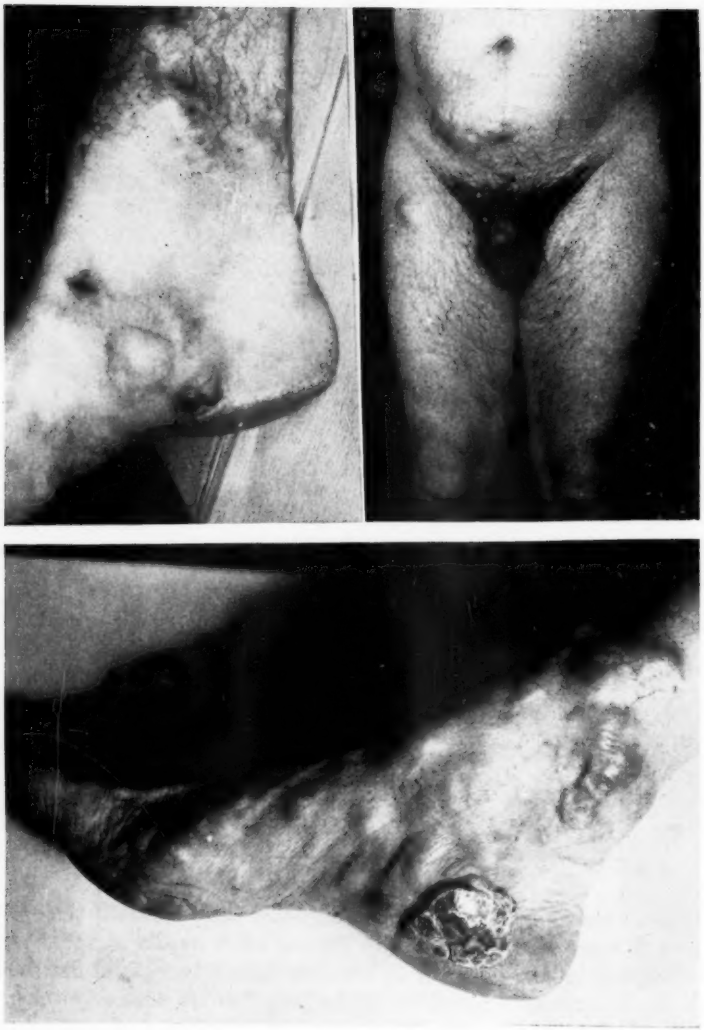

FIGURE 7. Photograph *(top)* shows the tumors present in 1937; patient was not seen again until January 1952, when he presented multiple tumors of the foot, leg, thigh, abdomen and scalp. He felt well, with no systemic complaints, returning only because he was concerned about the size of the growth on his heel *(bottom)*. The new tumors evidently are primary foci of Kaposi's sarcoma and not metastases.

nately, neither biopsy nor autopsy was performed so that a definite statement as to the cause of death in this case cannot be made.

From Table 4 it can be seen that the lower extremity was the predominant site of involvement, with the upper extremity next most frequently involved. A symmetric distribution was observed in most of the cases. Penile lesions were present in 2 patients; tumors were located on the ears in 2, on the face in 1 and on the scalp in 1. Six of the patients had plaques and nodules, 7 had nodules alone and 3 had plaques alone. In 2 cases tumors appeared in areas of purpura. In 1 the early lesions had the appearance of bullae, while in another the original diagnosis was granuloma pyogenicum. In 2 cases ulceration of the tumors developed.

Histopathologic studies were done in 13 of the subjects. In 2 the microscopic picture was interpreted as hemangioma (a subsequent biopsy in 1 of these was reported as fibroma) and in another 2 as fibrosarcoma, the remaining 9 being characteristic of Kaposi's sarcoma. The latter diagnosis was established for the 4 patients whose biopsies were histologically atypical, either by subsequent biopsies or by the clinical appearance of the lesions and the course of the disease. The 3 cases in which histologic investigation was not done were clinically typical of Kaposi's sarcoma.

Edema of an extremity was noted in 8 of the 16 cases. In 5 of these, edema preceded the appearance of cutaneous lesions; the diagnosis of varicose veins had been made in 2 and thrombophlebitis in 1 to explain the swelling.

TABLE 4

FEATURES OF LESIONS IN 16 CASES OF KAPOSI'S SARCOMA

CASE	LOCATION OF LESIONS	TYPES OF LESIONS	EDEMA	BIOPSY
1	Feet, toes, back	Plaques	Yes	Kaposi's sarcoma
2	Feet, hands, fingers, penis, ears	Bluish plaques, some ulcerated	Yes	Kaposi's sarcoma
3	Feet, toes, legs	Reddish and violaceous nodules and plaques	Yes	Kaposi's sarcoma
4	Feet, toes, legs, hands, penis	Violaceous nodules and plaques; purpura	Yes	Fibrosing hemangioma
5	Foot, arm	Nodules and bluish plaques	Yes	None
6	Foot, leg, thumb	Reddish nodules, some ulcerated	No	Fibrosarcoma; Kaposi's sarcoma
7	Feet	Nodules, some ulcerated	No	None
8	Feet	Bluish nodules	No	Kaposi's sarcoma
9	Feet, fingers, hands, arms	Reddish nodules	No	Kaposi's sarcoma
10	Feet, legs, hand, ear	Purplish nodules	No	Kaposi's sarcoma
11	Legs, hands, arms	Reddish nodules and plaques; purpura	No	Kaposi's sarcoma
12	Feet, toes, legs, hands, fingers, face	Plaques and nodules	Yes	Kaposi's sarcoma
13	Legs	Nodules	Yes	Fibrosarcoma; Kaposi's sarcoma
14	Feet, legs	Plaques and nodules	No	Kaposi's sarcoma
15	Foot, leg, thigh, abdomen, scalp	Nodules	No	Hemangioma; fibroma
16	Foot, arm	Plaques	Yes	None

Cytologic studies of the blood were done in 7 cases. Results of these examinations were essentially normal; no evidence of lymphocytosis, eosinophilia or monocytosis was found, and only 2 of the patients showed a mild anemia. In 3 cases a special search of the peripheral blood smear for the "abnormal mononucleated cells" of Flarer was made, but none were found.

In a recent report by Hurlbut and Lincoln[65] it was noted that 6 (46 per cent) of their 13 patients with Kaposi's sarcoma presented evidence of diabetes mellitus. Fischer and Cohen[53] reported a case of Kaposi's sarcoma of 25 years' duration which became complicated by the development of diabetes mellitus and lymphatic leukemia. They raised the question whether the diabetes in these patients might not be the result of development of tumor within the pancreas. Urinalyses were done in 11 of our cases and failed to reveal glycosuria; determination of the fasting blood sugar was made in 8 of these and each had a level within normal limits.

References cited will be included in the authors' reprints.

The Surgical Aspects of Jaundice

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Although the need for operative correction of the lesion in extrahepatic jaundice is urgent, it must be remembered that the most common cause of jaundice is medical, namely, virus hepatitis. Liver function tests are extremely valuable in differential diagnosis of these two conditions, but three or four tests must be performed to obtain a correct impression. Removal of stones in the common duct is curative and a very gratifying operation. Removal of carcinoma of the ampulla of Vater is followed by a five year survival of about 25 per cent, but following pancreatoduodenectomy for carcinoma of the head of the pancreas the five year survival is less than 5 per cent.

JAUNDICE is of two major types, intrahepatic and extrahepatic. The most common single cause of jaundice is virus hepatitis, in which there is damage to the liver cell and the liver is unable to excrete bilirubin. There may be an obstructive element in this hepatogenous jaundice because of edema which may compress the bile ducts.

WARREN H. COLE

Since differential diagnosis is so important in the management of patients with jaundice, a few remarks on the diagnosis of virus hepatitis are in order. Perhaps the most important symptom of virus hepatitis is weakness, which usually becomes manifest before the jaundice. Most patients have fever the first few days of the disease; almost all have anorexia; only a few experience vomiting; most have acholic stools for several days at the onset. In the mild form of the disease, of course, jaundice may not appear; in such a case the stools may not be acholic.

In obstructive jaundice the cause is extrahepatic, of course, and usually the common duct is involved. Various types of lesions producing obstructive jaundice include stones in the common duct, carcinoma of the ampulla of Vater, carcinoma of the pancreas, carcinoma of the terminal end of the common duct, and stricture. Another cause of obstructive jaundice is metastatic lesions in the liver.

Stones in the common duct are common, and an error in diagnosis is serious when they are present. The stone usually drops down from the gallbladder as a small stone through the cystic duct. It may stay there for weeks or months and enlarge, or it might pass on through. Many small stones will pass through into the duodenum, and when jaundice is instituted by stone it usually means that the stone is lodged in the terminal end of the common duct.

I would like to emphasize that jaundice is present in only slightly more than half the cases

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of common duct stones. Characteristically there is pain in the right upper quadrant with intermittent jaundice, and acholic stools of intermittent type. This pain is present in the right upper quadrant or in the epigastrium in perhaps 80 per cent of cases, and it is invariably most severe at the onset. We must be careful if we see these patients two or three weeks after onset, when they may have forgotten the pain which has diminished or disappeared.

One point about surgical intervention in obstruction due to stone: Obstructive jaundice itself does not produce hepatic insufficiency, but it will do so if infection occurs simultaneously or concomitantly, or if the obstruction is prolonged. Accordingly, in the average case, if the operation is not urgent because of suppurative cholangitis we will usually profit from several standpoints by waiting a few days. You can have more confirmation about the diagnosis, and thereby may eliminate the error of operating on a patient with virus hepatitis. Such an error may result in a death which could have been avoided by conservative treatment.

One condition which does demand emergency operation is suppurative cholangitis. When obstruction results in infection in the common duct, as may occur with stones in the common duct or even carcinoma of the head of the pancreas, operative intervention consisting of drainage of the common duct is essential. If the obstruction is due to a stone, the stone is removed.

A few points about indications for opening the common duct: If you feel a stone in the common duct or if there is a dilated, thickened wall of the duct, indications for opening it are strong; if there is a history of jaundice you must be very careful lest that jaundice may have been due to virus hepatitis. So the history of jaundice alone is not an indication for opening the common duct or even for operating on the patient until it can be proved to your reasonable satisfaction that the jaundice is obstructive in type.

Cholangiograms are helpful at the operating table, particularly if there is evidence of obstruction and no stone can be found in the common duct, or if several stones are found and you are concerned lest others be left.

We must also be certain at the operating table that the lead probe goes down through the sphincter of Oddi. One of the easiest ways, in my estimation, to tell whether or not the probe has passed through the sphincter is to press the probe against the wall of the duodenum; if you see the lead color of the probe, it is in the duodenum; if it is not in the duodenum but is pressing the posterior wall of the duodenum forward against the anterior, the thickness of the two walls is sufficient to prevent the lead color from showing through. That test seldom will fail.

I would estimate that the over-all mortality rate for choledochostomy for common duct stone is around 4 per cent. Glenn recently reported a mortality rate of 9.6 per cent in patients over 50 years of age and of 1.5 in those under 50. We don't fully appreciate the difference in mortality rate or in operative or physical reserve in elderly patients as compared with young persons. We are just beginning to appreciate that difference—that we must have these elderly persons in fine physical condition before we submit them to a major operation.

I would like to make a few remarks about carcinoma of the head of the pancreas and localized pancreatitis, because these conditions come into the picture very sharply in diagnosis. The jaundice present in a case of carcinoma of the head of the pancreas is apt to be of the painless type and the liver function tests are apt to be normal. The roentgenogram may help you, because occasionally it shows a deformity of the curvature of the duodenum. The stools are acholic at the onset, and once they become acholic they almost always remain so. Courvoisier's law is very helpful insofar as a large, dilated gallbladder is concerned, for it usually indicates carcinoma of the head of the pancreas.

The mortality rate is quite high following radical resection of the head of the pancreas and duodenum for carcinoma of the head of the pancreas. Kleinsasser and associates surveyed the literature and found that for 60 or 65 patients the mortality rate was between 25 and 30 per cent. That means we must expect

fairly good results before we submit these patients to such a drastic procedure. The five year survival rate is quite poor; I think there are only about 2 or 3 cases on record in which the patients survived five years following operation for carcinoma of the head of the pancreas.

These results should not be confused with those following resection for carcinoma of the ampulla of Vater, which is an entirely different lesion. Briefly the symptoms are similar except there is more pain, the jaundice and acholic stools are more likely to be intermittent, and blood usually is present in the stool because the lesion is ulcerating in the duodenum. The mortality rate following resection will be as high as that for carcinoma of the head of the pancreas, but the five year survival rate will be 20 or 25 per cent. Thus we must not fail to give these patients the benefit of resection if they have operable lesions.

The manifestations of localized pancreatitis in the head of the pancreas are similar to those of carcinoma of the head of the pancreas. This is not a common lesion, but every surgeon who has encountered it never forgets it because it is such a confusing lesion. The symptoms are very similar to those of carcinoma of the head of the pancreas and the lesion feels like carcinoma at the operating table. It is impossible to differentiate at operation between localized pancreatitis and carcinoma, unless carcinomatous nodules are present. Biopsy is not very helpful because if the tumor is located in the center of the mass the specimen probably will be taken from the superficial portion of the pancreas and show nothing more than mild pancreatitis. If you take the biopsy specimen deeply from the center of the mass you are apt to cut through a major duct.

Let's take the problem of stone in the common duct versus carcinoma of the head of the pancreas and apply this to our patients. The patient with stone in the common duct usually is over 40 years of age; the patient with carcinoma of the head of the pancreas usually is over 50. Stones are seen predominantly in women, carcinoma of the head of the pancreas in men. The onset is sudden with stone, and usually insidious with carcinoma of the head. Previous attacks usually have been present with stone. There is pain in more than 80 per cent of cases of stone in the common duct, and in less than 20 per cent of carcinoma of the head of the pancreas. Acholic stools are intermittent in stone; once they develop they are permanent in carcinoma of the head of the pancreas. There is a small shrunken gallbladder with stone and a large gallbladder with carcinoma of the head of the pancreas. Fever due to suppurative cholangitis may be present in either case.

A case which illustrates a very important point is that of a 67 year old woman with a three year history of jaundice. She had lost a lot of weight and was cachectic, and my first impression was that she had carcinoma of the terminal type. I learned long ago, however, that it is dangerous to make a positive diagnosis without operative confirmation, so a celiotomy was done. She had a fistula between the duodenum and gallbladder, where some

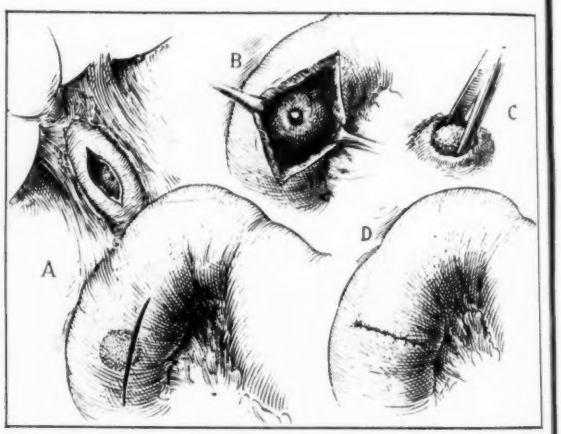

FIGURE 1. In this patient it was possible to palpate a stone in the common duct but not to remove it with a scoop inserted through an opening in the common duct. Since the stone was impacted in or near the ampulla of Vater the duodenum was incised and the stone was extracted through this approach.

stones apparently had ulcerated through into the gastrointestinal tract. One large stone was found in the common duct. We opened the duct and took out the stone, and she promptly recovered and has remained well for several years.

Another patient, an obese woman, had a two or three month history of jaundice. With a probe in the common duct I could feel an obstruction at the terminal end. I couldn't get a stone out, so I opened the duodenum. I still could feel a narrowing of the duct but could not get a stone out. I gave up in desperation after cutting the sphincter, and two months later a cholangiogram revealed a shadow almost typical of stone. Naturally I thought I had left a stone. Another cholangiogram was taken and this time the shadow was gone; two more and the shadow was still gone. It appears that the stone either dropped out or the shadow was an artifact. At any rate this woman is now well and we did not have to operate again. True, we gave her heavy bile salt therapy which chemically reduces the precipitation of cholesterol because of the increased salt in the bile. That may have had nothing to do with eradicating the stone, but I mention it because we have tried this method in 2 or 3 cases of residual stones in the common duct and have not had to reoperate to take out the stone. I don't wish to imply that this can be done in every case.

Several precautions must be taken in gallbladder surgery. Remember that three-fourths

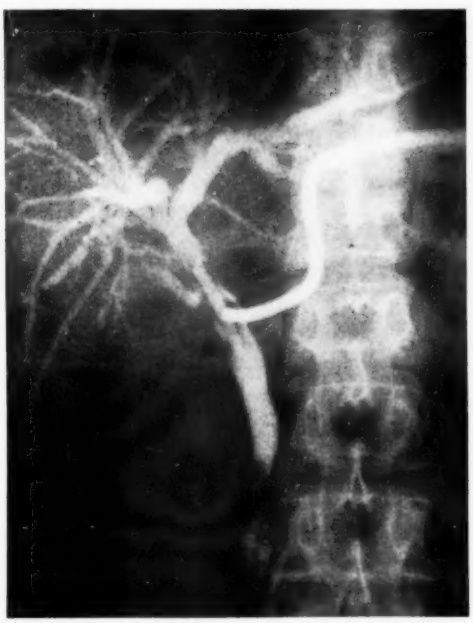

FIGURE 2. Cholangiogram taken three weeks after choledochostomy on an obese jaundiced woman. At operation, which was very difficult, a nodule was palpable in or near the pancreatic portion of the common duct. This nodule was thought to be a stone but even after 15 or 20 minutes' effort it could not be extracted. After discovery of the shadow by cholangiogram the patient was placed on daily bile salt therapy.

FIGURE 3. Cholangiogram taken one month after the one illustrated in Figure 2. The shadow is gone and was not seen in either of two additional cholangiograms taken several weeks later. The bile salt therapy which was given to increase the bile salt concentration may not have had anything to do with disappearance of the shadow, which presumably was a stone.

(From Cole, W. H.: Recent trends in gallbladder surgery. J.A.M.A. 150:631-637 [October 18] 1952.)

FIGURE 4. The barium meal in this patient revealed a greatly enlarged duodenal curve suggestive of cancer, which was corroborated at operation.

of the strictures of the common duct are due to trauma. A bleeding cystic artery might get loose, and if the bleeding is not controlled you may catch the common duct with your clamp; or you might lift up the gallbladder so much that the common duct is tented up, resulting in so much anatomic distortion that a clamp is placed on the common duct instead of on the cystic duct. You might try to get too much of the cystic duct stump out and, when tying it, pinch the common duct, with formation of a stricture later, particularly if infection develops around the ligature.

Cattell quoted some figures for the years 1942 to 1945 in a series of over 1000 cases in which he and others at the Lahey Clinic explored the common duct in 45 per cent; he found stones in 36.7 per cent of those explored and in 16 per cent of the total group. Contrast this with the lowest figure of 10.5 per cent explored by Glenn of New York who found stones in 69.1 per cent of those explored and in 7.2 per cent of the total group. The symptomatic results may not be as different as they appear, although the percentage of explored ducts in Glenn's series is much lower than in Cattell's. The explanation probably lies in the fact that many of the ducts explored in Cattell's series contained small stones which would have passed spontaneously on down through the common duct into the duodenum.

Presentation of Patients

A 63 year old white man was admitted to the medical service on October 14 with com-

FIGURE 5. This patient had a large malignant ulcer (4 by 5 cm.) at the sphincter of Oddi. There was insufficient deformity to diagnose the lesion by x-ray, although the arrow points to a suggestive lesion.

plaints of vague and mild epigastric pain, jaundice, weakness, anorexia and weight loss, all these symptoms having become progressively severe for approximately two weeks. This is an instance of a rather sudden onset of jaundice without much pain. Examination showed the abdomen to be rounded in contour with a few dilated veins. There was an epigastric mass extending five fingerbreadths below the right costal margin. He had a moderate anemia and an icterus index of 112. Total protein was slightly lowered (6.5 gm.), but the alkaline phosphatase was greatly elevated (38.5 units) and pointed to obstructive jaundice. Cephalin flocculation in 24 and 48 hours was normal; thymol turbidity was 1.0. A barium enema disclosed a moderate-sized filling defect in the cecum. His stools were acholic and remained so.

In this case there was no evidence of damage to the liver cells, but the findings indicated obstructive jaundice. At laparotomy an anaplastic lesion was found in the cecum along with some metastatic lesions in the liver, explaining the jaundice. He has lost weight and has a cachectic appearance supporting the presence of a metastatic carcinoma.

The normal liver function tests indicated that the jaundice was obstructive in type, but naturally did not indicate which type of lesion was producing the jaundice. Operation confirmed the diagnosis, but the obstructive lesion was in the liver and was not correctible.

The second patient, a 39 year old man, was admitted on October 31 with a seven day history of dull pain in the left side of his chest. His symptoms were relatively mild and of short duration. The liver was palpable. The stools were brown, and he had an icterus index of 37 and a serum bilirubin of 2.9, both of which indicate definite but moderate jaundice. Cholesterol esters were only 15 mg. per hundred cubic centimeters, and total cholesterol was 99 mg., both indicating hepatic disease. Total protein was 4.7 gm. per hundred cubic centimeters with an albumin-globulin ratio of 1:47, strongly indicating hepatic disease. The thymol turbidity test was normal and the alkaline phosphatase test result was 16.3 pointing to an obstructive element in the jaundice.

These figures indicate that more than one liver function test must be done if we are going to rely on them for diagnosis; about four are necessary before a specific interpretation of liver function can be made.

In this case there is evidence of hepatic disease which is supported by the acholic stool. The most likely diagnosis is cirrhosis. Since the liver is enlarged, we would perhaps call it hypertrophic in type. I do not have time to go into the argument concerning the question of enlargement of the liver preceding atrophic cirrhosis, nor will I go into treatment in this case, because it is medical. Therapy for cirrhosis of the liver is extremely unsatisfactory.

Aphasia—Etiology and Classification

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A simple and practical classification of the aphasic disorders encountered in clinical practice emphasizes the so-called receptive aphasias because these are often due to emboli, infections and tumors. Their accurate recognition may reveal that they are not always manifestations of simple apoplexy and may call for specific and effective treatment.

APHASIA, a word introduced in 1861 by Trousseau, is a broad inclusive term which simply means an acquired inability to communicate by signs or language.

LOUIS J. KARNOSH

In the subhuman brain, such as that of the ape, there is no evidence of much language function as we understand it, and experimental work on the monkey has contributed little or nothing to our knowledge of speech as it exists in man. Language in its full flower is a distinctly human attribute, and we know how it works only from careful clinicopathologic correlations. This is how Broca studied it when he discovered his well known motor speech area. In his enthusiasm over his findings he exuberantly declared that when all the physiology of speech is revealed, the whole physiology of the psyche will be exposed and the days of the psychologist and the psychiatrist will come to an end.

Being a neurologist, his was the folly of oversimplification. However, later experience with the subject of aphasia has indicated that simplification in this matter has definite and practical value. The overelaborate expositions of Henry Head[1] are cumbersome, and few have the stamina to read through his 2 heavy volumes studded with new and unfamiliar terms, while the abstruse psychodynamic descriptions of Goldstein[2] affect one like a fine but heady wine.

The much simpler classifications of Weisenburg[3] and Nielsen,[4] which cling to some anatomic basis, are far more plausible and of easiest clinical application.

The most unique feature of all language function in the human brain is its lateralization or one-sidedness—its restriction for the most part to one hemisphere. Nothing like this has been demonstrated in animals, and perhaps it is this element alone which explains man's supremacy in self-expression.

This lopsidedness of speech function is dramatically demonstrated in a daring ablation where the right hemisphere has been removed in large part from patients with brain tumor. From studies made of these persons by neurosurgeons and neurologists, the evidence is clear that the capacity to carry on all the major functions of speech—that is, to understand the spoken word, to read, to comprehend meaning, to answer correctly and to write—is not impaired as long as the domi-

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nant hemisphere remains intact. Not a few of these people continue to thrive and lead normal lives except for being handicapped by a spastic left hemiplegia.

Weisenburg[3] made a practical classification when he divided all aphasia into that which is receptive, that which is expressive, that which is a mixture of the two and, finally, that which is amnesic.

By receptive aphasia he means all those disturbances which were first described by Wernicke and which hinder the perception, recognition and meaning coming to the ear, the eye or the fine sensations in the hand.

By expressive aphasia is meant the inability to convey meaning to others through word of mouth or by writing and other sign languages.

Let us proceed with a simple but useful subdivision of receptive aphasia. The first subtype is that defect in which there is a failure to understand the spoken word, called auditory agnosia. The lesion is usually in the first 2 temporal convolutions. Such a patient has lost the meaning of both word and sentence structure and his answer is usually a senseless jargon, which he continues to produce not knowing that it is meaningless. However, he does retain his normal inflections, his own peculiar accents and other idiosyncrasies which are his natural characteristics, for these emotional elements of speech come either from deeper centers or possibly from the opposite hemisphere.

A second receptive aphasia is the loss of the ability to understand or recognize the meaning of the written or printed word, denominated as word blindness or alexia. Here the patient in ordinary conversation is entirely normal but finds himself utterly baffled when confronted by printing or handwriting; to him it is a strange mixture of letters and nothing more. The patient can recognize objects or pictures of objects, but their symbols written on paper mean nothing.

A third type of aphasia is probably the most intriguing of all. As mentioned before, Weisenburg called it amnesic aphasia but it is best known as anomic aphasia. Here the patient shows no apparent defect in spontaneous conversation, understands and responds in an entirely normal manner. However, when suddenly confronted with a simple object such as a pen, a watch or some less common article, the patient cannot quickly bring out of memory the name of the object—that is, he cannot quickly associate the image of the thing with its vocal symbol. Yet at the same time he makes it clear by spontaneous conversation that he knows its meaning and use. Should, moreover, the name of the object be presented to him on the blackboard along with 20 or more other names of objects, he will unerringly select the correct one. Or, should the examiner vocalize a list of several objects, including the correct one, the patient can again make a correct selection. In short, the patient with anomic aphasia needs the visual or oral stimulus of the symbol in order to recognize the correct name.

To recognize these 3 receptive aphasias, namely, word deafness, word blindness and anomic aphasia, is all-important in diagnosis. We know that the lesions in all 3 are always in the occipitotemporal area, and generally near or in the cortex of that region. They are too often erroneously called motor aphasia. Be on guard if a patient has some speech block or cannot read and at the same time shows no evidence of typical apoplexy with hemiplegia. Few of these patients show any form of paralysis of either limbs or face, but most of them will show some degree of visual field change such as a quadrant or a complete hemianopsia. This is sure proof that the lesion is in the occipital and posterior temporal areas and not in the internal capsule.

Why is this diagnostically important? Because these unique aphasias are rarely due to a simple stroke. They are accidents more likely due to rarer lesions such as emboli, infections or simple blood clots. Emboli coming into the cerebral circulation from the heart walls or from elsewhere cannot get into the internal capsule; they spread out over the cortex, leaving in their wake patches of degeneration. Such emboli may be those of septicemia, those of subacute bacterial endocarditis and, most of all, the detached blood clots in an auricular fibrillation. Less com-

mon are metastatic cancer cells.

In brief, of some 40 cases of receptive aphasias, less than half were due to cerebral arteriosclerosis, while 60 per cent revealed such entities as subacute bacterial endocarditis and multiple embolism and 25 per cent turned out to be brain tumors. These revelations about receptive aphasia make it clear that proper diagnosis is often a critical issue in these conditions which are often amenable to adequate treatment.

Only a few comments are necessary about Broca's expressive aphasia. In the first place it is an extremely rare condition but is consistently confused with a common condition. True expressive aphasia is a complex defect in which the patient has complete comprehension of what he wishes to convey in speech but is unable to assemble the proper combination of muscle patterns in the motor cortex to produce the desired words. He can move tongue, lips and vocal cords at will, but the words refuse to emerge correctly. It is like the pianist who can hit each individual key but has lost the ability to produce chords. Again, in this motor aphasia there may be no neurologic defect in face or limbs.

While motor aphasia is rare, that which it closely resembles is the most common speech disturbance in clinical experience—namely, the disturbed articulation which goes along with the common variety of right-sided stroke. This is not an aphasia at all because it means no impairment in language integration itself; it is merely a defect in articulation where the tongue is weak, the lips are paretic and the pharyngeal muscles on one side are immobilized. This is known as dysarthria and should not be designated as any form or degree of true aphasia.

Case Report

We have at the Cleveland Clinic a striking case of receptive aphasia in a patient who is 65 years old and who had signs of congestive failure for about a year prior to the first study made in June 1952. This condition was announced by dyspnea, palpitation, ankle edema and hepatic engorgement. He had a mitral insufficiency. After this examination he improved following the administration of digitalis.

Two weeks later he awoke one morning with numbness on the right side of the body and incoordination of the right arm and leg. He was hospitalized and found to have no true hemiplegia; he appeared alert, was quick to answer questions and was able to carry on normal conversation. He was erroneously labelled a case of slight right hemiplegic stroke. Someone had noted that he had trouble cooperating in the visual field test, but the most significant feature of his story is that not until September 12, nearly 3 months later, was it realized that he could not read and that he had a complete right hemianopsia.

He had some anomic aphasia, but his main handicap was his inability to make sense out of printed and written material. It was a source of frustration not to be able to read the headlines in connection with the election.

This reading trouble is not all due to the hemianopsia which still exists. He can see extremely well, and he can recognize some isolated letters, but the quick recognition of words as total images is impossible. He cannot write from dictation, but he can duplicate a written word—which is simple mimicry, for he can rarely recognize the word he copies. Obviously, this man's language functions are all intact except those which depend on recognition of the written symbols of language.

What is the pathology? Looking back on the story, we now conclude that on the critical night when he awoke with numbness of the right side, he had a multiple embolism of the entire posterior left hemisphere which left a rather permanent residue in the angular gyrus.

It is our hope and belief that other intact areas will take over and some of his alexic troubles will be relieved after adequate retraining and practice.

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Diagnosis of Multiple Sclerosis

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Much can be done from a therapeutic standpoint to arrest or retard the progress of multiple sclerosis, and distressing symptoms can be relieved. Many contractures and other disabilities can be avoided through physical therapy and simple orthopedic appliances. The earlier the diagnosis is made and treatment instituted, the better the chance to control exacerbations and prolong remissions.

HINTON D. JONEZ

THE most prevalent neurologic condition we face today is multiple sclerosis. This disease takes a number of forms, but the diagnosis is not difficult if a careful study of the symptoms is made. Typical multiple sclerosis is characterized by periods of exacerbation and remission. However, absence of these periods does not rule out the diagnosis, as the initial lesions may be so extensive and overwhelming that it is not possible for a true remission to occur. Typical multiple sclerosis produces symptoms involving all parts of the central nervous system.

Charcot in about 1868 gave this disease the name multiple sclerosis and described the classic triad of scanning speech, nystagmus and intention tremor as diagnostic of the condition. Today we realize that this triad is present only in advanced cases. The earlier symptoms sometimes are fleeting, such as stiffness and spasticity of a limb for a day or two, or diplopia that may cause the patient to consult an ophthalmologist. There may be other bizarre neurologic symptoms that are overlooked at the time, but as the patient's condition becomes more serious with later attacks, the picture of multiple sclerosis unfolds itself.

The majority of persons with multiple sclerosis have been misdiagnosed as being neurotic or sufferers from "nerves" during their earlier attacks. Brickner[1] observed: "Often a patient with multiple sclerosis, if pressed, may be able to recall a fleeting symptom years before, such as numbness of the fingers which disturbed his writing for perhaps an afternoon. On a basis of these mild, vague and transitory symptoms one is not justified in making a diagnosis of multiple sclerosis, but a prognosis of this disease should be considered." Concerning prognosis, Lichtenstein[2] said: "The prognosis in multiple sclerosis is not as grave as many would have the profession believe. Some individuals have two or more mild attacks and never go on to develop the advanced form of the disease."

As we view the disease today, the cardinal signs and symptoms are as follows:

1. Increased deep tendon reflexes.
2. Nystagmus.

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FIGURE 1. Positive Babinski reflex in multiple sclerosis.

FIGURE 2. Abdominal reflexes usually absent in multiple sclerosis.

3. Ataxic tremor of upper extremities.
4. Loss of abdominal reflexes.
5. Spastic, ataxic or spastic-ataxic gait and station.
6. Spasticity of one or both upper or lower extremities.
7. Positive Babinski sign.
8. Speech disturbances.
9. Pallor of temporal halves of the optic disks.
10. Diplopia with ocular palsies.
11. Urinary incontinence.
12. Remissions leading to disappearance of some or all of the symptoms.

Very few patients, of course, present all these symptoms. However, a diagnosis is justified if four or five are present involving three parts of the central nervous system. Figures 1 through 8 illustrate some of these findings.

Differential Diagnosis

Before making a definite diagnosis of multiple sclerosis, care must be taken to rule out space-displacing neoplasms of the brain and cord, herniation of intervertebral disks, especially in the cervical region and, of course, syphilis. Hereditary ataxia and muscular atrophies frequently are mistaken for multiple sclerosis. To illustrate this, I call your attention to Table 1, which represents 2000 con-

FIGURE 3 *(left)*. Past-pointing and intention tremor in typical multiple sclerosis.

FIGURE 4 *(center)*. Ankle clonus in typical multiple sclerosis.

FIGURE 5 *(right)*. Tripod cane-walking by an ataxic-spastic multiple sclerosis patient.

secutive patients who were accepted at our clinic. Most of these patients came to us with a definite diagnosis of multiple sclerosis made at a reputable general clinic or by an accredited neurologist. On further study and follow-up these cases fell into the various diagnostic categories listed.

In our opinion the percentage as represented by the various demyelinating diseases in this table probably represents to a certain degree the prevalence of these conditions throughout the country. The table shows that multiple sclerosis is not the killer it is supposed to be. This bears out the finding of a study made in a large midwestern clinic to the effect that the life expectancy of the patient with multiple sclerosis is near that of a normal person.[3] Under the heading of multiple sclerosis these atypical types were included: hemiplegic, tabetic, spastic paraplegic, sacral, cervical, bulbar, pontine and cerebellar.

Lateral sclerosis—Many neurologists today dispute the separate clinical entity of lateral sclerosis. Older clinicians spoke of both primary lateral sclerosis or spastic paraplegia and posterior lateral sclerosis or ataxic paraplegia. The ataxic paraplegia is analogous to Friedreich's ataxia. Most cases of lateral sclerosis are diagnosed as multiple sclerosis. Wechsler[4] maintains that lateral sclerosis is

TABLE 1

SUMMARY OF 2000 CONSECUTIVE CASES PREVIOUSLY DIAGNOSED AS MULTIPLE SCLEROSIS AND RECLASSIFIED AT ST. JOSEPH HOSPITAL, MULTIPLE SCLEROSIS CLINIC, DURING THE PAST FIVE YEARS

RECLASSIFICATION	TOTAL CASES		DEATHS	
	Number	*Per Cent*	*Number*	*Per Cent*
Multiple sclerosis				
Typical	1548	77.4	41	2.65
Atypical	49	2.45		
Lateral	233	11.65	9	4.07
Familial	51	2.5	1	1.96
TOTAL	1881	93.0	51	2.65
Amyotrophic lateral sclerosis	51	2.5	15	29.61
Familial ataxia				
Friedreich's	17	0.85	1	5.88
Marie's	21	1.5		
TOTAL	38	2.35	1	2.63
Charcot-Marie-Tooth disease	10	0.15		
Miscellaneous				
Combined sclerosis	3	0.15		
Pseudosclerosis of Westfall	2	0.1		
Pseudobulbar palsy	1	0.05		
Transverse myelitis	1	0.05		
Undetermined	11	0.55		
Determined by autopsy				
Brain tumor	1	0.05	1	100.0
Internal hydrocephalus	1	0.05	1	100.0
TOTAL CASES	2000			

FIGURE 6 *(left)*. Spastic scissoring gait in multiple sclerosis (lateral type).

FIGURE 7 *(center)*. Ocular palsy in multiple sclerosis.

FIGURE 8 *(right)*. Exaggerated patellar reflex (all tendon reflexes usually increased in multiple sclerosis).

definitely a separate condition, but acknowledges that a certain percentage of these cases eventually do become multiple sclerosis. There should be a wait of several years before the possibility of multiple sclerosis is ruled out. Lateral sclerosis is a disease of the pyramidal tract involving first the lower extremities, but it may ultimately involve the upper extremities. Its course is very slow and the symptoms are much milder than those of the usual typical case of multiple sclerosis. The vast majority of clinicians today diagnose all these cases as multiple sclerosis.

Familial multiple sclerosis—Familial multiple sclerosis has been a disputed question since Charcot's day. In 1930 Goldflam[5] stated that "the familial occurrence of multiple sclerosis is not a rarity and indicates the significance of endogenous factors in many cases." In 1950 Mackay[6] stated: "At present it is certainly true that familial cases occur much more commonly than is generally assumed, although the modern textbooks minimize or ignore the subject." Early in our study we noted the occurrence of this condition in related persons more frequently than would be expected according to the ordinary incidence. In our study of 2000 cases the well known familial diseases were excluded and observations were limited to cases of multiple sclerosis alone. Those cases classed as being familial were those in which the disease was present in at least two members of the patient's immediate family (grandparents, parents, brothers and sisters) or in at least three

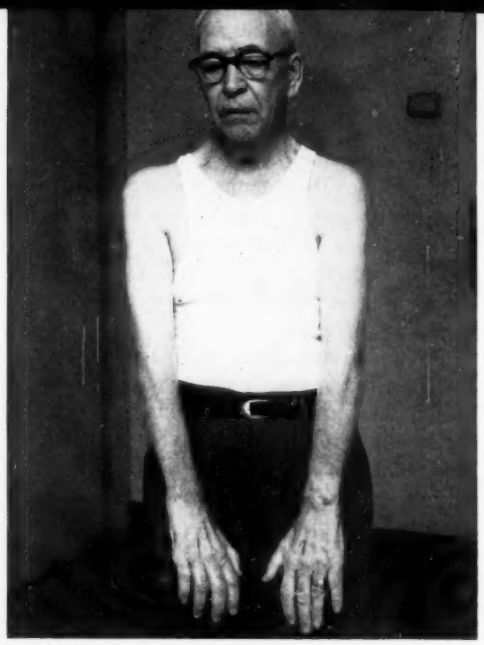

FIGURE 9 *(above)*. Atrophy of upper extremities in amyotrophic lateral sclerosis.

FIGURE 10 *(left)*. Typical foot in Friedreich's ataxia.

FIGURE 11 *(below)*. Ataxic gait of patient with Marie's ataxia.

members of the rest of the family (uncles, aunts or cousins). As shown in the table, our findings are surprising in that the familial incidence was high, and this seems to bear out the contention that there is a familial vulnerability or a definite familial type of multiple sclerosis.

Amyotrophic lateral sclerosis—Amyotrophic lateral sclerosis frequently is misdiagnosed as multiple sclerosis, especially early in the disease. There should be no difficulty in differentiating between these two after a short time, as amyotrophic lateral sclerosis usually is rapid in its progress. Most authorities consider it a combination of progressive muscular atrophy and lateral sclerosis, and not in the multiple sclerosis classification (Figure 9). As can be seen in the table, the mortality rate is very high in this condition.

Ataxias—Friedreich's ataxia and Marie's ataxia are slowly progressive, familial, heredodegenerative variants of the same disease. Friedreich's ataxia begins in childhood, usually between the ages of 7 and 9 years, and involves the spinocerebellar tracts, while Marie's ataxia occurs after the age of 20 years and is mainly cerebellar. Both frequently are mistaken for multiple sclerosis. Usually the Friedreich's foot or toe (Figure 10) and the markedly ataxic gait in Marie's ataxia (Figure 11) will differentiate these two conditions from multiple sclerosis.

Charcot-Marie-Tooth disease—Peroneal muscular atrophy is commonly called Charcot-Marie-Tooth disease. While occasionally mistaken for multiple sclerosis, the differential diagnosis should not be difficult because the disease slowly causes the lower legs to atrophy. The calves and lower thighs are affected, with the resulting term "stork legs and champagne bottle thighs" (Figure 12). There is a marked steppage gait (Figure 13). The disease is heredodegenerative in character. It usually begins in young adult life, with later involvement of the upper extremities.

FIGURE 12 *(left)*. "Stork legs" in Charcot-Marie-Tooth disease (peroneal muscular atrophy).

FIGURE 13 *(below)*. Steppage gait in Charcot - Marie-Tooth disease.

Etiology

The multiple sclerosis syndrome constitutes over 90 per cent of demyelinating conditions. Etiologic theories are many, among

them vitamin or mineral deficiencies, virus infections, emotional instability and glandular disturbances.[7] During the last decade there has been a growing acceptance of the idea that allergy plays a major role in the etiology of this disease.[8-25]

Treatment

All the diseases listed in Table 1 should be considered chronic and incurable. Nevertheless symptomatic treatment is advantageous in all, as in most chronic conditions. In our clinic, allergy management plays the major role in treatment of patients with multiple sclerosis.[26] All are given scratch tests, and all keep diet diaries. We have found that those with severe and multiple food allergies are the most spastic cases, whereas those with strong allergies to molds are next in severity. Pollenosis appears to cause the seasonal exacerbations. Geographically the dairying and cereal-producing areas present the largest number of patients from a population ratio standpoint. We give allergenic extracts and prescribe diets to all patients as indicated.

Histamine is used as a hyposensitizing medium. It also plays its part as a vasodilator. Vasodilatation is advised by all clinicians who treat patients with this disease. Among the more common vasodilators are nicotinic acid, ETAMON CHLORIDE® and DICUMAROL®. However, histamine given by any method is the most powerful intracranial vasodilator known.[27]

Physical therapy is necessary for correction of deformities and gaits. Relaxing agents are helpful as an adjunct. We use PROSTIGMIN®, MYANESIN® and d-tubocurarine in a repository menstruum of oil and wax. In our opinion d-tubocurarine is the most useful in the greatest number of cases.[28, 29]

Adenosine monophosphate and adenosine triphosphate have been very valuable in controlling bladder symptoms. Gonadotrophic hormones are employed as indicated for emotional instability. Psychotherapy[30] plays a major role in elevating the mood and the morale. Antidepressants benefit some patients.[31]

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Diagnostic Problems Encountered in Thoracic Diseases

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Diagnostic problems in thoracic diseases are not always easy. Roentgenologic study of the thorax is by far the most accurate single examination available at present, but even this procedure has its restrictions. It is safer at present to explore an indeterminate thoracic lesion than to observe it. It is not always safe to watch a lesion until the results of gastric culture for fungi and tuberculosis become available.

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In diseases of the thorax, one must be constantly on guard in the manner of assessment of the various available diagnostic aids so that too hasty conclusions may not be reached on the basis of one or two positive findings. These findings may be in error and thus an accurate diagnosis may be delayed. In final analysis, any error in diagnosis is the responsibility of the clinician, for he alone has access to all the facts about the patient. He should be qualified to judge the accuracy of the various tests that he orders. For this reason it is well to take inventory of the most common procedures used in diagnosis of thoracic diseases to determine how much reliance can be placed on them.

Physical diagnosis of disease in the thorax is important, yet it has great limitations. Many patients who have advanced pulmonary tuberculosis may present no abnormalities on physical examination. Conversely, physical findings may be misinterpreted and an erroneous diagnosis may be made. Some years ago I attempted to study the records of all patients who had postoperative atelectasis during a one year period at the Mayo Clinic. It was found that in some instances the diagnosis had been made on physical examination alone. The findings described were usually reduced breath sounds over the right lower lobe. It soon became apparent that for the purpose of accuracy this group of patients must be excluded from the study. Interest was centered on the physical findings that could be expected in all patients who had had operations on the biliary tract. Surprisingly, most such patients had variable abnormal physical findings in spite of the fact that they were making an uneventful recovery from operation. The finding of reduced breath sounds over the right lower lobe was common. Bronchial breathing over the right lower lobe could be detected at times during the early phases of the postoperative period. It was considered that compression of the lobe by upward retraction on the undersurface of the liver was probably the factor that pro-

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duced these transient findings. In practically all cases, roentgenograms of the thorax showed nothing abnormal. It became apparent that atelectasis probably did not exist if it could not be shown roentgenologically.

Roentgenologic study of the thorax is by far the most accurate single examination available at present for all thoracic lesions. Even this procedure has its restrictions, which must be recognized. Rigler,[1] in writing about these limitations, divided the problem into two parts: (1) roentgenologic findings that lead to the detection of an abnormal process and (2) findings that lead to identification of the nature of the lesion. He pointed out that findings at the earliest stages of the disease may identify the nature of the lesion. It is for the clinician, however, to make certain that all findings fit the picture in order that an accurate diagnosis can be made.

Rigler emphasized that thoracic roentgenograms have their greatest value in discovering an unsuspected lesion. In a specific problem, the limitations of roentgenologic examination as a diagnostic method must be realized. Rigler stated that in pulmonary tuberculosis the lesion can be seen early in its development, well before the onset of symptoms or physical signs. However, a latent period exists before roentgenologic studies give evidence of disease; this period may be as long as 8 to 20 weeks after a person is infected with tuberculosis. The roentgenogram may be normal even though an extensive tuberculous process is present. More actual involvement is always present in active tuberculosis than can be demonstrated by roentgenologic study. In miliary tuberculosis, the roentgenologic findings follow the onset of symptoms; 1 to 7 weeks may be necessary before shadows show in the roentgenogram.

Rigler considered that metastatic lesions of the lung usually must reach a size of 3 mm. before they can produce roentgenologic changes. The fact that a primary carcinoma of the lung has to reach a certain size and density before it can be recognized as an abnormality makes it possible for metastasis to occur before the primary tumor can be recognized roentgenologically. My colleagues and I have seen metastatic brain lesions produce symptoms before the primary bronchogenic carcinoma could be demonstrated roentgenographically.

Garland[2] has studied the problem in a different way. He pointed to the fact that an element of uncertainty is present in all aspects of medical diagnosis in which physicians are asked to give an objective or a subjective opinion.

In order to investigate the relative diagnostic efficiency of various roentgenographic and photofluorographic technics used in mass surveys, a board of roentgenology was appointed in 1944. This board was made up of 2 roentgenologists and 3 specialists in diseases of the thorax; the services of a biostatistician were available. Each member of the board made independent interpretations of four sets of films and found to his astonishment that not only did he differ from his colleagues in apparently simple problems but he even differed with himself in the interpretation of a significant number of the same films reviewed on two separate occasions. The board recommended that this phenomenon be subjected to extensive detailed investigation.

In a review of the various methods used in mass roentgenologic surveys of the thorax, it was observed that a significant incidence was present of both overreading and underreading. Films interpreted by a majority of reviewers as showing no apparent abnormalities by all known criteria were interpreted as abnormal by some persons (overreading, or false positives). Conversely, some films accepted by a majority as showing evidence of disease were interpreted as showing nothing abnormal by other reviewers (underreading, or misses). Furthermore, the same reviewer was inconsistent with himself in a surprising number of instances on both scores when the same films were interpreted on two separate occasions. These errors were attributed initially to technic, to the presence of small lesions and to "the human equation." They were measured by a study of the relative frequency with which a reviewer was inconsistent with others and with himself on two separate evaluations of the same set of films. The rea-

sons for missing diagnoses are the size, location and density of the lesion and the "attitude" of the observer.

Errors present in other tests also are evident. At present, cytologic study of sputum at the clinic discloses malignant cells in approximately 70 per cent of all cases in which carcinoma of the bronchus is present. In approximately 2 per cent of all tests giving positive results, these results are "false positives."[3] It is possible to secure positive results in bronchoscopic biopsy in only 48 per cent of bronchogenic carcinomas.

Some error is inherent in examination by direct smears of sputum for acid-fast organisms. In only about 40 per cent of instances will organisms be obtained from the sputum of patients who are producing tubercle bacilli from the tracheobronchial tree.

If cultures of gastric washings for *Mycobacterium tuberculosis* are done on three separate occasions, it is estimated that almost all patients who have "open" pulmonary tuberculosis can be detected. Positive results of a gastric culture present a great degree of reliability. At the clinic, a virulence test is performed on organisms obtained from the first gastric culture that shows positive results; the chance of the results of this gastric culture being falsely positive is only 0.3 per cent.[4]

Errors of clinical judgment in thoracic disease are of course the most important of all. Certain roentgenograms may appear to show a lesion in the lungs, the appearance of which actually is caused by such factors as nipple shadows or tumors in the breast or thoracic wall. Pneumonia may simulate carcinoma of the bronchus or active tuberculosis. Roentgenograms taken a week after the original film of the thorax may appear normal in the former.

Tuberculosis may simulate cancer, and cancer may look like tuberculosis; these circumstances should make the clinician be on his guard and insist on proving the origin of every lesion to the best of his ability.

Errors in judgment as to when surgical exploration of the lung should be done are important. In general, it is safer at present to explore an indeterminate lesion than to observe it. It is not always safe to watch a lesion until the results of gastric cultures for fungi and tuberculosis become available.

Thus it can be concluded that diagnostic problems in thoracic diseases are not always easy. The clinician should insist that all findings in a given problem fit the clinical picture. It is only in this way that diagnostic errors can be minimized.

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CORRECTION

On page 391 (May 1953 issue) of the article by Dr. Alec Horwitz entitled "The role of surgery in peptic ulcer," the second sentence of the second paragraph should have read:

"The hypersecretion of acid-gastric juice is four times greater in duodenal ulcer."

Intractable Exophthalmos in Thyroid Disease

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The incidence of progressive exophthalmos in cases of thyroid disease and the pathologic changes that initiate the condition are discussed. Results of bilateral orbital decompression in 78 cases are reported, and the technic of the procedure is outlined.

JAMES L. POPPEN

PROGRESSIVE exophthalmos that advances to such a degree that the patient's sight is jeopardized and perhaps the eyeballs are lost fortunately is a rare complication of thyroid disease. It does occur with sufficient frequency, however, so that its importance should be stressed. Close cooperation of the internist, ophthalmologist and surgeon is essential, because occasions arise that make the decision as to the proper sequence of treatment difficult.

At the Lahey Clinic approximately 60 per cent of the patients with primary hyperthyroidism have varying degrees of exophthalmos. Cattell found that in 21 per cent the exophthalmos persisted following thyroidectomy, but in only slightly over 1 per cent did the degree increase. It is this latter group of patients with which we are concerned. Fortunately in only 1 in 1000 patients with primary hyperthyroidism successfully treated for the thyrotoxicosis will the degree of exophthalmos progress to such an extent that orbital decompression is desirable. It must be emphasized that primary hyperthyroidism is not a requisite for so-called malignant exophthalmos; it may also occur in patients who have chronic thyroiditis or toxic adenomas.

Seventy-eight patients with thyroid disease have been subjected to bilateral orbital decompression at the clinic. Forty-nine were women and 29 were men. Their ages ranged from 26 to 67 years.

There are many theories concerning the pathologic changes that initiate exophthalmos, especially progressive exophthalmos in thyroid disease. No one factor, however, can definitely be considered the sole cause. In all probability the fluid balance in the tissues plays a part. Because of the unusual bony space occupied by the orbital tissues it can readily be understood that increase of fluids in these tissues would increase the volume of the space-filling tissues. Adequate room for this increased space-occupying tissue can be obtained only by the forward displacement of the eyeball. The effect on the blood and lymph flow is dependent on the amount of increase of the fluids in the tissue. Certainly the venous flow would most likely be interfered with first, resulting in stasis and further swelling and producing a vicious circle. This, however,

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does not explain several of the findings at orbital decompression.

Most of us know that a localized, expansile, intradural lesion in the spinal canal creates space first by decrease in the available fluid space, then by distention of the dura and subsequently by absorption of the extradural fatty tissue. It could be assumed that a similar process would take place in the orbital tissues. It is possible to predict to a reasonable degree the amount of fatty tissue present in the orbit by the degree of exophthalmos and the associated increase in resistance of the retro-orbital tissues. In patients who have a high degree of exophthalmos but a minimal amount of chemosis, the fatty content is normal or possibly greater than normal owing to the myxedematous changes that have taken place in the fatty tissues themselves, and the muscle tissue is relatively normal, especially if the extraocular movements are unchanged. On the other hand, in the patient who has a moderate to a high degree of exophthalmos with marked chemosis and edema of the eyelids and a phlegmonous type of firmness in the retro-orbital tissues, the orbital contents are filled only with gray, thickened, firm, enlarged muscle tissue and very little fat. Why this occurs is difficult to understand, unless the fibrous tissue that tends to keep the eyeball in place and prevents it from protruding is more highly developed in some patients than in others. In the patients in whom most of the fatty tissue has been absorbed and the muscles individually have greatly enlarged, a great degree of extraocular muscle palsy usually is found.

We do not believe that the rapidity with which the thyrotoxicosis is controlled has any relation to the progress of exophthalmos. Exophthalmos occurs in patients who have had no form of treatment, who have recently had a thyroidectomy, who have had x-ray treatment 10 years before the onset of progressive exophthalmos, and who have had treatment with radioactive iodine.

Myxedema, if present, varies so greatly in degree that E. C. Bartels is of the opinion that it plays no part in the etiology of progressive exophthalmos. If present, it should be treated. The basal metabolic rate may be low, normal or high, and progressive exophthalmos is therefore not singly related to metabolism.

Orbital decompression should be considered whenever changes in vision, extraocular muscle palsies and exposure keratitis become evident. Cosmetic surgery is indicated only when the patient's livelihood is in jeopardy or the psychic trauma is such that life becomes unbearable. The patient who has marked retro-orbital resistance with only moderate exophthalmos may be in a more precarious situation than the patient with a high degree of exophthalmos in whom retro-orbital resistance is only slightly increased. In the former, marked chemosis and lid edema are present, associated with multiple extraocular muscle palsies, or there may be scotomas of various sizes and involving various parts of the visual fields, which may become so large as to cause virtual blindness. We have encountered cases in which scotomas involve the inferior quadrants bilaterally, the upper quadrants bilaterally (either in the nasal or temporal field), or the entire lower half or upper half of the visual fields. The muscle groups most frequently involved are the elevators, the upper gaze being most frequently impaired. Marked pallor of the head of the optic nerve is occasionally seen, with associated reduction in vision. This probably is due to the increase in the bulk of tissue in a very confined region (the annulus of Zinn) in which very little fatty tissue is present, thus permitting very little self decompression by absorption of fat. The increased pressure in this constricted area causes direct pressure on the optic nerve and interferes with the blood supply to the nervous tissue itself.

The technic that we employ has been described previously and is much the same as that used by Naffziger, with the exception of the extent of the decompression and the type of bone flaps. In the preoperative preparation of the patient, chemotherapy is instituted 12 to 24 hours before surgical intervention. Roentgenograms of the skull are taken to determine the size of the perinasal sinuses, particular attention being given to the frontal orbital and the orbital ethmoid sinuses. Many times these sinuses extend over the greater

portion of the roof of the orbit and they must necessarily be removed or entered to obtain a satisfactory decompression. Whenever this is necessary it is important to save as much of the mucous membrane as possible so that it may be inverted and folded on itself as it is gently displaced into the larger sinus spaces. The opening in the mucous membrane heals readily and seals itself in a few hours. The exposed areas are protected with GELFOAM® and as the orbital fascia is incised, it may be reflected over the open sinus to insure a firm closure. To prevent extraocular muscle palsies following surgery we studiously avoid opening the annulus of Zinn. It is perhaps unnecessary to unroof the optic nerve head even if choking or pallor of the optic disk is present. I have unroofed the optic nerve in all patients in whom optic pallor or marked choking of the optic disk has been demonstrated. In all probability this procedure is not necessary if the annulus of Zinn has been adequately decompressed.

It has been our practice in the past to use a two man team for the orbital decompression. More recently, however, the time element was not increased when a single team was utilized, the entire procedure, including the preoperative temporary canthorrhaphy and bilateral orbital decompression, taking usually from one and one-half to two hours. It is our practice to insert two 8 gauge rubber catheters, one in each orbital space, and it is surprising how much sanguineous fluid escapes even though the operative site was perfectly dry at the time of closure. The burr openings are filled with tantalum buttons and the coronal incision sutured with a single layer of through-and-through black silk stitches. Two thin-rubber ventricular bags, partially inflated with air, are placed over the closed eyelids and reasonably firm pressure is applied by means of an ACE® bandage. The pressure should be released within four hours after operation and then replaced with slightly less pressure exerted by the ace bandage. Dressings are changed twice a day and the catheters are removed within 12 to 24 hours after the surgical procedure. The pressure over the eyes is continued for seven days. At each dressing the patient is questioned concerning perception of light through the closed eyelids. After the third or the fourth day, heat is applied to the eyes for 20 minutes three times a day, pressure bandages being applied between the heat treatments. The temporary mattress sutures employed to close the eyelids are removed at the end of the seventh day.

Definitely diminished vision varying from mild reduction in visual acuity to almost complete blindness was noted in slightly more than 51 per cent of the patients before surgery. In 12 per cent vision diminished temporarily after operation; however, within a few weeks vision improved markedly over that present before operation. In 10 per cent visual acuity improved very little, if any. This was especially true of the patients who had only a slight change in visual acuity before surgery. It is in this group that I must, much to my regret, report that 1 patient who had a complete quadrantic defect and whose vision was 20/100 became completely blind following operation. The surgical procedure in this case was particularly simple. Optic pallor was present before surgery and the patient had a fairly high degree of myopia. No pressure was exerted on the optic nerve at any time during the operative procedure. Pressure was exerted over the orbits in the usual manner postoperatively. The patient was completely blind when the lid sutures were removed. The pupils did not react to light, there was considerable edema of the retina with periarterial streaking of blood along the retinal vessels, and the optic nerve heads appeared as they did before operation. I do not know the exact cause of this catastrophe. I believe a contributing factor, or possibly an important factor, may be the pressure exerted on the orbits by the air-inflated bags since we all know that patients with high myopia are particularly vulnerable to changes in the retina. Certainly in patients who have a high degree of myopia, great caution should be used so far as any external pressure on the eyeball is concerned.

There have been no operative deaths in this series. In 2 of our early cases in which the larger bone flaps were used, temporary frontal lobe signs appeared. There have been

3 subsequent deaths: 1 from coronary thrombosis, 1 from a diabetic coma and the other of unknown cause. It is important to emphasize that the degree of exophthalmos and the degree of extraocular muscle palsies may improve up to seven years following orbital decompression, as demonstrated by several of our cases.

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MEETINGS AND POSTGRADUATE COURSES

American Institute of Dental Medicine: Tenth annual meeting, October 25-29, at the Desert Inn and El Mirador, Palm Springs, California. Applications and full information may be secured by writing Miss Marion G. Lewis, Executive Secretary, 2240 Channing Way, Berkeley 4, California.

Boston University School of Medicine and Massachusetts Memorial Hospitals: Postgraduate course of clinical lectures and demonstrations for practicing physicians, Wednesdays, 2:00 to 4:00 P.M., November 11, 1953 to April 28, 1954, Dr. Chester S. Keefer, Director. Modern diagnostic methods and treatment will be stressed. For further information and details of registration, write: Dr. James M. Faulkner, Dean, Boston University School of Medicine, 80 East Concord Street, Boston 18, Massachusetts.

Second International Congress of Cardiology, September 12-15, 1954, Washington, D.C. The congress will be followed by the annual session of the American Heart Association. Program will be printed in French, Spanish and English, and immediate translation will be made of some discussions. A series of postcongressional visits to leading cardiac clinics in the United States and Canada has been arranged. L. W. Gorham, Secretary-General, 44 East 23rd Street, New York 10, New York.

Pan American Medical Association: Three year scholarships will be given annually to 2 physicians in each of the 22 nations in the Western Hemisphere. The 3 year resident postgraduate studies are to be the equivalent of those of the diplomates of the various American boards. At the end of 3 years there will be 132 physicians taking the courses, and then each year 44 will be sent back to their countries to practice and teach. Executive Director, Dr. Joseph Jordan Eller, 745 Fifth Avenue, New York, New York.

University of Minnesota, Minneapolis: Continuation course in radiation therapy for specialists, October 26 to 31, 1953, at Center for Continuation Study. Guest faculty will include Dr. Robert McWhirter, Department of Radiotherapy, Royal Infirmary, Edinburgh; Dr. Raymond Zirkle, Institute of Radiobiology and Biophysics, University of Chicago; Dr. Edith Quimby, Department of Radiology, Columbia University, New York; and Dr. Lauren V. Ackerman, Professor of Surgical Pathology and Pathology, Washington University School of Medicine, St. Louis. Course will be directed by Doctors Leo G. Rigler, Professor and Head, Department of Radiology, and K. W. Stenstrom, Director of Radiation Therapy.

Continuation course in dental problems in medicine for physicians, October 21, 1953, at Center for Continuation Study. Subject matter will include examination of the teeth, gums and mouth; oral manifestations of systemic disease; management of injuries involving the mouth and jaws; afflictions of the temporomandibular joint; and the fluoride problem. Faculty will be members of the faculties of the University of Minnesota Dental and Medical Schools.

Symposium on cardiovascular physiology and surgery, September 14-16, in the Museum of Natural History Auditorium. Presented under the auspices of the Minnesota Heart Association, the symposium will be open, without tuition fee, to all physicians and to qualified investigators in the field of cardiac physiology. Further information may be obtained from the Director, Department of Continuation Medical Education, University of Minnesota Hospitals, Minneapolis 14, Minnesota.

The Endocrine Society and the University of Minnesota announce a Postgraduate Assembly in Endocrinology and Metabolism for Physicians, the sixth annual assembly sponsored by the Society, to be presented September 28-October 3 in the Museum of Natural History Auditorium and the Center for Continuation Study. The program will be presented under the direction of Dr. Edward C. Reifenstein, Jr. and Dr. C. J. Watson. Chairman of local arrangements is Dr. Edmund B. Flink.

GENERAL PRACTICE *Review*

Medical Management of Acute and Chronic Bronchitis and Bronchiectasis; Progress Review

This department offers the latest, proved methods for the treatment and management of commonly encountered conditions based on the extensive experience of the physicians who prepare these summaries for POSTGRADUATE MEDICINE.

ALVAN L. BARACH*

College of Physicians and Surgeons, Columbia University, New York City

Acute Bronchitis

ALVAN L. BARACH

ACUTE bronchitis may be caused by infectious or irritant chemical and physical agents. The majority of cases follow the common cold. The extensive investigations of Andrewes[1] of England confirmed previous studies of Dochez[2] and others[3,4] that coryza is a viral disease, contagious and communicated by ordinary human contact. The patient with a head cold may spread virus-laden secretions over food, nearby individuals and, in fact, over the greater part of an ordinary room. Influenza virus A, B, or any of the other strains may also produce acute inflammation of the tracheobronchial tree. Acute bronchitis is uniformly present in measles and whooping cough, the latter disease being due to *Hemophilus pertussis*. Other infections which may be complicated by acute bronchitis include typhoid fever, diphtheria, trichinosis, scarlet fever and primary atypical or virus pneumonitis.

When the common pyogenic organisms are responsible for pharyngitis, laryngitis and bronchitis, the acute inflammatory process may progress to the alveoli with development of a frank pneumonia. The organisms most frequently isolated are the *Pneumococcus*, especially types 1, 2, 3, 5 and 7, the hemolytic *Streptococcus*, *Klebsiella pneumoniae*, *Staphylococcus aureus* and *H. influenzae*. Infections of the bronchi with *Candida albicans*, occasionally encountered in infants and debilitated adults, have been observed more recently following prolonged treatment with the broad spectrum antibiotics or as a result of multiantibiotic therapy such as combinations of penicillin and streptomycin.

Predisposing causes of infection, especially in the very young and the very old, include chilling, fatigue and pre-existing chronic sinus or bronchopulmonary disease.

The preventive treatment of the disease consists mainly of attempts to limit exposure to individuals who have acute coryza; washing the hands and face would appear to be a reasonable procedure after contact with in-

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fected persons. Avoidance of cold, damp weather and chilling is an additional precaution especially indicated in patients with chronic sinus or bronchopulmonary disease.

A mild attack of bronchitis occurring in an otherwise healthy individual generally subsides spontaneously in 3 to 10 days. When constitutional symptoms are present, such as fever and malaise, rest in bed is indicated until the temperature has been normal for 24 hours. To allay an irritant dry cough, codeine, 0.015 to 0.03 gm., prescribed at four hour intervals, is the drug of choice. In many patients, especially those with a history of allergy, aminophylline administered on an empty stomach in a dosage of 0.2 gm. by mouth two to three times daily is more effective than cough suppressants. Some of the newer preparations of aminophylline contain benzocaine (cardalin), an antinausea factor which makes possible the administration of larger oral doses, such as 0.35 gm. on retiring and on arising. In patients with more severe bronchospasm, aminophylline may be administered rectally or, very slowly, intravenously; in many of these cases, inhalation of nebulized bronchodilator aerosols, epinephrine, racemic epinephrine (VAPONEFRIN®) or ISUPREL® (1-[3′, 4′-dihydroxyphenyl]-2-isopropylaminoethanol hydrochloride) results in prompt symptomatic benefit.

Of the expectorant drugs, potassium iodide, 5 to 10 drops of a saturated solution three times daily, is especially useful in patients with bronchial asthma or pulmonary emphysema. The addition of Fowler's solution (tincture of arsenic), 2 to 4 drops three times daily, is at times remarkably effective in stopping or eliminating bronchospasm, when administered for two or three weeks. In children in whom bronchial mucus is not easily expectorated, the use of ipecac in doses to produce vomiting is often valuable. Mustard plasters, flaxseed poultices and steam inhalations, with or without compound tincture of benzoin (1 gm. per pint), are now rarely employed except in the young.

Antibiotic therapy is indicated in cases in which a suppurative process is manifested by persistence of purulent sputum. Since the virus of the common cold is not influenced by any antibiotic agent, treatment should not be started until there is a clear indication of a pyogenic infection. In patients with chronic respiratory disease the desirability of prompt intervention with appropriate antibiotic agents is unquestionable, since exacerbation of the underlying condition generally takes place if this is not done. Penicillin was the drug of choice in treatment of infections caused by gram-positive bacteria, in the absence of known sensitivity. Effective blood levels are obtained by oral administration of 500,000 to 1,000,000 units on an empty stomach twice daily or intramuscular injection of 500,000 to 1,000,000 units of procaine penicillin. However, the increasing incidence of allergic reactions and resistant staphylococci is limiting its use.

Sulfadiazine may also be given for these infections. The broad spectrum antibiotics, especially terramycin and AUREOMYCIN®, are also potent against pyogenic infections. The dosage required appears to be less than that ordinarily used, 1 gm. daily in four divided doses being adequate in most cases. Acidophilus cultures either in milk or in tablet form are recommended to reduce the development of *Monilia* in the colon. Terramycin and aureomycin are indicated in the treatment of *K. pneumoniae* infections, with or without streptomycin, but carbomycin and erythromycin should be used in infections with gram-positive bacteria.

Chronic Bronchitis

Chronic bronchitis is characterized by a long-standing inflammation of the bronchi and their subdivisions, with, in most cases, fibrotic and atrophic changes in the submucosa. Although the disease may occur as a separate clinical entity, it is most commonly associated with other chronic respiratory illnesses.

Chronic bronchitis may result from repeated exposure to the previously described causes of the acute form, namely, infection, allergic reaction or contact with physical and chemical irritants. Although the disease may follow frequent attacks of acute bronchitis, it often develops, especially in older people, either with-

out clearly recognized cause or in association with a chronic illness of the respiratory tract such as pulmonary emphysema, pulmonary fibrosis, bronchiectasis, pneumoconiosis or sinusitis. The condition is generally bilateral unless local inflammation is the result of foreign body pressure from an enlarged tracheobronchial lymph gland or an aneurysm of the aorta.

Cough and expectoration are characteristic of the uncomplicated case of chronic bronchitis. Sputum may be either scanty, viscid and tenacious or mucoid and more copious, especially in cases with allergic reaction to such allergens as pollens and house dust, or in patients with bacterial allergy resulting from a sensitizing reaction to the micro-organisms involved. When expectoration is purulent or mucopurulent, pyogenic organisms are generally found on smear and culture. In some cases both infectious and allergic factors are present, giving rise to an abundant mucoid, viscid sputum with purulent plugs found at the bottom of the container. In the uncomplicated case, fever and systemic symptoms are absent except during exacerbation of the infectious process.

When chronic bronchitis is associated with other respiratory disease such as pulmonary emphysema, pulmonary fibrosis, bronchial asthma or pulmonary tuberculosis, as well as various entities produced by long-continued inhalation of irritant inorganic dusts, the symptoms of the associated diseases then dominate the clinical picture, frequently resulting in disturbances of respiratory function and body chemistry, the more common manifestations being dyspnea and wheezing respiration, accompanied in severe cases by anoxia and increased levels of carbon dioxide in the blood.

The diagnosis of chronic bronchitis can be established only when the wide variety of respiratory and cardiac conditions which produce cough and expectoration are excluded. Under such circumstances a patient in good general health whose complaint is limited to chronic cough may be considered to be suffering from chronic bronchitis. Other conditions which may simulate or be associated with chronic bronchitis include left ventricular failure, pulmonary tuberculosis, carcinoma of the bronchus, bronchiectasis, lung abscess, aortic aneurysm, foreign body in the bronchus, pulmonary emphysema and the various pulmonary fibroses.

A differential diagnosis between bronchiectasis and chronic bronchitis is securely established only with the aid of LIPIODOL® bronchograms. Patients with chronic cough and mucoid expectoration, with or without asthmatic symptoms, are more apt to have chronic bronchitis than are those in whom the expectoration is repeatedly found to be purulent.

The so-called "cigaret cough" should not be accepted as a diagnosis. Even though symptoms of cough and expectoration are relieved by cessation of smoking, this does not rule out the presence of an underlying disease process. Any irritant, even cold air, may act as a trigger mechanism to precipitate coughing in various types of cardiorespiratory illness. Furthermore, patients with left ventricular failure and bronchial asthma may cease to cough as a result of cigaret smoking when compensation has been restored and bronchospasm terminated.

Treatment has only recently emerged from the stage of palliative therapy—use of cough suppressant and expectorant drugs such as codeine, potassium iodide and ammonium chloride. If adequate facilities are available for diagnosis and controlled therapy, many of the patients can be benefited considerably.

In cases where physical or chemical agents play a role, proper control of the environment is obviously indicated. The inhalant allergens, such as feathers, dust and animal dander, may cause chronic cough and expectoration; bronchospasm and cough may also be produced by allergic reaction to bacterial infection. Hyposensitization programs, including the use of antigens such as dust and bacteria, have been helpful at times. Symptomatic treatment of cough in such cases includes the use of ephedrine and aminophylline by mouth or, in more severe cases, aminophylline intravenously or rectally.

In the presence of asthmatic symptoms the

inhalation of nebulized epinephrine aerosols is likely to produce symptomatic relief of dyspnea and expectoration of bronchial secretions. Repeated paroxysms of coughing may be terminated in some cases or may be controlled by the administration of a drug such as DEMEROL®, 50 mg. given four times daily for four or five days.

In patients whose chronic bronchitis is the result of infection, opportunity for remarkable improvement may be present, especially in cases with coexisting pulmonary emphysema or other serious respiratory disease.[5] A definitive diagnosis must first be made by smear of the sputum with demonstration of pus cells, isolation of the predominant organisms and a test of their sensitivity to the antibiotics available. In contrast to the bacterial flora described in the section on acute bronchitis, *Staph. aureus* is the most common invader in cases of chronic bronchitis. Unfortunately, both hemolytic *Staph. aureus* and *Staph. albus* have developed in recent years a progressive resistance to antibiotics. In in vitro studies, approximately 60 per cent of the strains encountered were found to be resistant to penicillin and 25 per cent resistant to terramycin and aureomycin.[6] Although only 17 per cent of these organisms have developed resistance to chloramphenicol, the hazard of adverse effects on bone marrow has caused limitation of the use of this drug except in cases unresponsive to other antibiotic therapy. Additional organisms isolated on sputum culture include nonhemolytic streptococci, pneumococci, *K. pneumoniae*, *H. influenzae* and, at times, *C. albicans*, *Proteus* and *Pseudomonas aeruginosa*, secondary invaders which have become invasive when prolonged broad spectrum or multiple antibiotic therapy has eliminated the common bacterial flora of the lungs.[7-11]

Therapeutic management of these cases presents many difficulties. For infections due to gram-positive organisms, penicillin is the drug of choice unless the patient has become allergic to it or the organisms themselves have become resistant. Since the *Staphylococcus* is the only micro-organism invading the respiratory tract that has developed resistance to penicillin, the newer drugs, MAGNAMYCIN® and ILOTYCIN®, may be used instead of penicillin, or the broad spectrum antibiotics may be employed for periods of 5 to 10 days in the hope of arresting the infection or restoring the organism's sensitivity to penicillin. The diaminoethyliodide ester of penicillin may be effective, since concentrations of this drug higher than those of benzopenicillin are found in the lungs and in the sputum.* If a highly resistant strain of *Staph. aureus* is the cause of chronic bronchopulmonary infection, inhalation of penicillin aerosol in dosage of 1,000,000 units daily may be required to eliminate this organism. When improvement has been accomplished by the use of penicillin, this drug should be continued as long as the sputum is mucoid, despite the appearance of gram-negative organisms on culture of the expectoration. Treatment may be carried on for one or two months with penicillin, either by mouth or aerosol, to obtain a prolonged arrest of chronic bronchial infection.

In cases in which the sputum becomes purulent *during* penicillin administration, either because of the development of penicillinase or through infection with gram-negative organisms such as the *K. pneumoniae*, terramycin or aureomycin may be administered, with or without streptomycin, for five to eight days or until the sputum becomes mucoid again. CHLOROMYCETIN® has been used in a variety of cases resistant to other antibiotics, including *Staph. aureus* and *albus*, *Proteus* and paracolon bacilli.

*When the proofs of this paper were received, the Council on Pharmacy and Chemistry reported that "14 severe anaphylactic reactions including 3 that resulted in death have followed the administration of penethamate hydriodide (neo-penil)" (J.A.M.A. 151:1105, 1953). Since we have recently encountered 4 instances of acute, severe reaction to intramuscular injection of neo-penil, the use of this drug appears to be accompanied by certain hazards, in the dosage in which it has been employed. In 3 cases of bronchial asthma or pulmonary emphysema with an associated chronic bronchitis or bronchiectasis, the intramuscular injection of 1,000,000 units was followed within a period of less than a minute by loss of consciousness and muscular twitching or convulsive movements of the upper extremities, without bronchospasm or dyspnea, the symptoms receding within five minutes. In the fourth patient, an intramuscular injection of 2,000,000 units was followed by pallor, rapid pulse and faintness without complete loss of consciousness. Since this patient previously had received injections of 500,000 units without ill effects, succeeding injections of 500,000 units and 1,000,000 units were given for one and six days respectively without adverse manifestations. The clinical response of these patients suggests a toxic rather than an anaphylactic-like shock reaction to the drug and that its therapeutic use should be reinvestigated with a smaller dosage, such as 200,000 units four times daily, instead of the larger single injections of 1,000,000 units.

In the event that *Proteus*, *Pseudomonas* and *C. albicans* should grow on sputum culture during prolonged treatment with broad spectrum antibiotics or multiple antibiotic therapy, namely, streptomycin and penicillin, all antibiotic drugs should be stopped until these organisms disappear. Less risk of invasion by these bacteria, which are ordinarily contaminants, is to be expected with increasing skill in the application of antibiotics. For example, the dosage of the broad spectrum antibiotics originally recommended was approximately 2.0 gm. daily. As indicated by the studies of Habif,[12] much smaller amounts of terramycin, even as little as 250 mg. twice daily, have been found therapeutically effective in surgical infections; with these smaller dosages and the routine use of cultures of acidophilus organisms ingested with milk, the incidence of diarrhea and the development of monilial organisms in the stool appear to have been markedly reduced.

The treatment of acute exacerbations of infections in patients with chronic bronchitis, and of such associated pulmonary diseases as bronchial asthma and pulmonary emphysema, is of considerable importance. The principle of therapy should be eradication of the microorganism considered responsible for the disease by a chemotherapeutic or antibiotic agent which has a limited but effective spectrum, such as penicillin, sulfadiazine, magnamycin or ilotycin, rather than the initial use of broad spectrum antibiotics. The latter agents are best reserved for organisms unresponsive to penicillin, such as *K. pneumoniae*. Alternate use of different antibiotics as indicated in the individual case is preferable to multiple antibiotic therapy, especially when prolonged treatment is planned.

As the hazard of invasion by resistant organisms has grown clearer, methods for accomplishing drainage of bronchial secretions have assumed increased importance. These include postural drainage, employment of the head-down position in bed at night, deliberate coughing after inhalation of bronchodilator aerosols, the use of bronchodilator drugs such as aminophylline rather than suppressants such as codeine and, in cases with an allergic disposition, the use of potassium iodide to promote a freer flow of less viscid mucus.

The recent introduction of mechanical means of simulating the human cough has resulted in the development of a portable exsufflator by which, in many instances, retained secretions are effectively eliminated from the lungs.[13,14] This device operates by means of full inflation of the lungs followed by sudden application of negative pressure during the expiratory cycle. The resultant flow rates are higher than those of the maximally vigorous cough of patients with bronchial asthma and pulmonary emphysema.

As noted, too much emphasis may be placed on the harmful effects of coughing, such as that due to the effect of cigaret smoking, and too little value on coughing as a means of cleansing the lungs of retained mucopurulent sputum.[15] Antibiotic therapy is more apt to provide prolonged relief if it is accompanied by conscientious measures to promote bronchial drainage.

Bronchiectasis

Multilobar bronchiectasis may occur either as a separate clinical entity or, more commonly, in association with other chronic respiratory diseases such as bronchial asthma, pulmonary emphysema, pulmonary fibrosis, pneumoconiosis and pulmonary tuberculosis. The chronicity of symptoms in this illness is largely related to retention of infected bronchial secretions. Impairment of ciliary action of the mucous membranes and of peristaltic contraction of the muscle of the affected bronchi has long been known. More recently, studies indicating that the bronchial wall is at times collapsed by a relative increase in pressure of the surrounding alveoli revealed the ineffective nature of the cough in patients with defective lung elasticity.[16-18] In some cases this premature collapse of the bronchi during cough is aggravated by varying degrees of bronchospasm. Furthermore, bronchospirometric studies have shown that, at times, the diseased lung segment has a tidal volume only one-fourth that of the normal lung.[19] Physiologic abnormalities such as irregular and impaired alveolar ventilation have

been recognized as factors impairing bronchial drainage,[21,22] especially in patients with pulmonary emphysema.

The bacteria responsible for infection are similar to those found in cases of chronic bronchitis. In communities in which penicillin is extensively used, the *Pneumococcus* disappears except in instances of acute respiratory infection or in patients in whom the disease has been followed by lung abscess and pneumonia or emphysema. Destructive changes in the bronchi, although generally the result of infection, may also be caused by the inhalation of chemical agents.

Various theories have been advanced to explain in part the pathogenesis of bronchiectasis on the basis of physical pressures due to coughing, accumulation of bronchial secretions, bronchial obstruction, atelectasis and traction on the bronchi as a consequence of pulmonary or pleural fibrosis. According to Lisa and Rosenblatt,[35] mechanical factors are seldom of importance, even as contributing causes. The inflammatory necrotizing process is said to be, in itself, enough to produce ectasia. Bronchial obstruction and atelectasis may produce their most significant effect by the impairment of drainage. Paranasal sinusitis as a cause of bronchiectasis is also doubtful and, in fact, a reverse relationship may be present, namely, sinusitis as a complication of bronchiectasis. Bronchiectasis has been noted following influenza, whooping cough, measles, scarlet fever, cystic fibrosis of the pancreas, and as a complication of vitamin A and D deficiency.

Bronchial drainage, long recognized as an aid in the treatment of bronchiectasis, has assumed increased importance with the development of physical aids for the elimination of bronchial secretions. Exsufflation, especially when combined with negative pressure, has resulted in the elimination of retained mucopurulent sputum from the lungs of patients with chronic pulmonary disease whose previous coughing had failed to produce expectoration.[13,14,23] The mechanism of exsufflation depends on full inflation of the lungs by a pressure of 35 to 40 mm. of mercury followed by abrupt termination of the pressure, which permits the lungs to deflate rapidly without opposition from gradually decreasing intrabronchial pressure. Exsufflation with negative pressure is produced by connecting the patient's mouth or face with the suction side of a negative pressure blower when peak inspiratory pressure has been reached. In this way a large tidal volume of air, 1500 to 2500 cc., is moved out of the lungs at a velocity 30 to 50 per cent higher than that achieved by patients with pulmonary disease by their maximal cough. The apparatus illustrated in Figure 1 was developed by Barach, Beck and Smith for intermittent positive or negative pressure

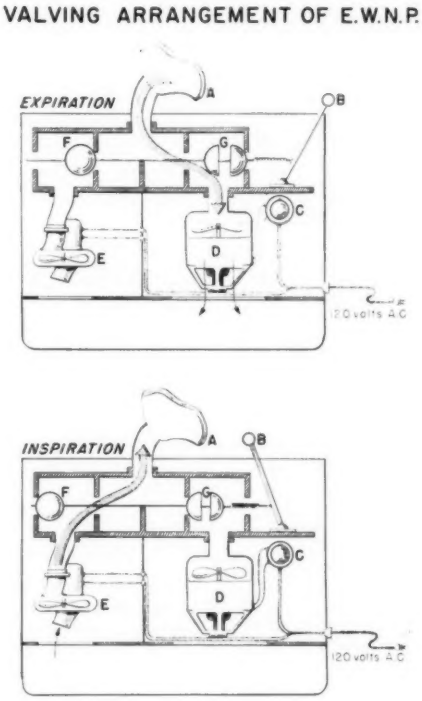


FIGURE 1. During inspiration the mask (A) is connected to a pressure blower (E) by which inflation of the lungs is established. The duration of inspiration, generally 2.5 seconds, is regulated by maintaining closure of compartment (G), either manually or by a solenoid. Pulling the lever (B) connects the mask with the negative pressure blower (D) by closing the valve (F). The Variac (C) regulates the flow rate and negative pressure used. The peak positive pressure may also be controlled by a valve connected to the positive pressure airway, not shown in this diagram.

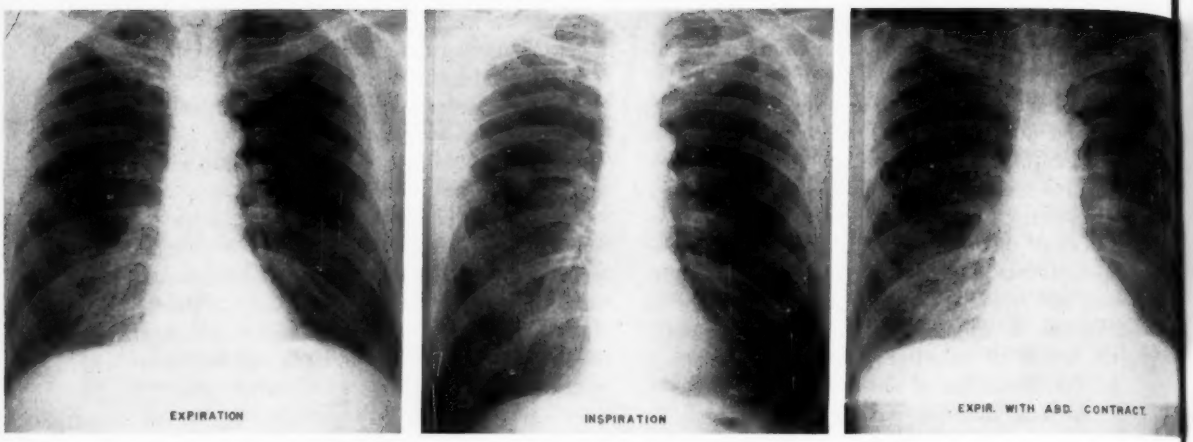


FIGURE 2. DIAPHRAGMATIC MOVEMENT AFTER BREATHING EXERCISES IN A PATIENT WITH CHRONIC BRONCHITIS AND PULMONARY EMPHYSEMA. Over-aeration of both upper lobes seen in x-ray. In the lower lobes, bronchial and vacular markings are more well-defined, indicating the importance of ventilating these segments of the lungs by training in diaphragmatic respiration. The films were taken with the patient standing and wearing a low abdominal belt. The center film reveals deep inspiration. The increase in expiratory emptying of the lungs that may be obtained by vigorous contraction of the abdominal musculature is shown, as well as the more readily learned use of the diaphragm and elastic recoil of the lungs in expiration, without employment of the abdominal muscles.

breathing and resuscitation.

Postural drainage is valuable not only by the conventional method of leaning over the edge of the bed but also by adoption of a resting bed position in which the foot of the bed is tilted upward at an angle of 16 degrees. Drainage may thus be attained during periods of repose by day and during sleep at night. Another physiologic advantage of the head-down position in bed, especially for patients with impaired lung elasticity, is the initiation of diaphragmatic breathing by upward pressure of the viscera on the diaphragm; better alveolar ventilation of the bases of the lungs produces a physiologic state in which secretions are more efficiently eliminated in subsequent spontaneous coughing.

Breathing exercises which promote diaphragmatic breathing, including such procedures as manual compression of the lower chest and upper abdomen, also aid in the promotion of bronchial drainage.[26] The degree of diaphragmatic excursion that may be developed in cases of pulmonary emphysema is shown in Figure 2.

Relief of bronchial obstruction due to spasm of the bronchi is of considerable importance in the large number of patients in whom varying degrees of asthmatic breathing are found. Aminophylline and ephedrine may be administered by mouth and nebulized bronchodilator epinephrine compounds may be inhaled either in concentrated solution such as 1 per cent epinephrine, 2.25 per cent racemic epinephrine (vaponefrin) or isopropyl adrenalin (isuprel), or in atropine-sodium-nitrate bronchodilator compounds.* Continuous nebulization of bronchodilator substances in more dilute solutions with or without 1 per cent NEOSYNEPHRINE® may be employed using either oxygen or a pump as a source of pressure.[27,28]

Methods of combining intermittent pressure breathing and inhalation of bronchodilator aerosols have been employed with specially designed apparatus by which oxygen is gen-

*In the older literature concerning the treatment of asthma, inhalation of sprays of sodium nitrate and atropine were commonly employed. In a study of these compounds in bronchial and cardiac asthma, Marder and the author have used the following solution:

Atropine	4 gr.
Sodium nitrate	14 gr.
Propylene glycol	15 gr.
Aqua distillata q.s. ad	½ oz.

The inhalation of 2 to 5 drops of this mixture frequently results in marked relief of bronchospasm with a corresponding increase in vital capacity

erally inhaled at a peak inspiratory pressure of 12 to 15 cm. of water with simultaneous inhalation of the bronchodilator aerosol.[29-31]

In addition to its use with exsufflation, expiratory negative-pressure breathing after a full spontaneous inspiration has also been employed as a method of decreasing pulmonary distention in cases of pulmonary emphysema as well as to facilitate expectoration of retained secretions.[5,25]

The importance of facilitating drainage in patients with bronchiectasis has been illuminated by our increasing knowledge of the hazards as well as the benefits of sustained treatment with the more powerful antibiotic drugs. Since new infections with organisms not previously pathogenic may result from continued multiple antibiotic therapy or use of the broad spectrum drugs which eliminate the common gram-positive and gram-negative organisms from the respiratory tract, careful precautionary laboratory control by sputum cultures is indicated in the employment of the more powerful antibiotic drugs.

The problem of sterilizing the bronchi of a patient in whom a pint of purulent secretion is retained in the lung is obviously more difficult than that in the case of a patient in whom effective bronchial drainage has been obtained by the measures already outlined. Although bronchoscopic aspiration in itself is of clear value, the necessity for daily repeated bronchial drainage makes the use of this method less feasible than mechanical exsufflation with negative pressure, especially by means of the portable device and other measures cited. Some of the principles of antibiotic therapy in chronic bronchial disease have been discussed in the section on treatment of chronic bronchitis, as well as in more detail elsewhere.[10]

Certain salient points may be emphasized as follows:

1. Infections caused by gram-positive organisms may be treated with penicillin unless the patient has become allergic to it. In cases of resistant *Staph. aureus* infection, inhalation of aerosol penicillin in high dosage has been found effective when parenterally administered penicillin is ineffective. Magnamycin and ilotycin appear to be highly effective agents with a limited spectrum against gram-positive bacteria, including resistant staphylococci.[36] Since allergic reactions have not yet been noted, these drugs may, to a considerable extent, replace penicillin as well as the broad spectrum antibiotics.

2. The use of the broad spectrum antibiotics such as aureomycin and terramycin is reserved for those patients who have become refractory to penicillin as a result either of the development of penicillinase or of infection with gram-negative organisms, especially *K. pneumoniae*. Chloromycetin is used in those patients who are unresponsive to other antibiotic drugs, and is at times of crucial value. No adverse effects of this drug have been noted in our series of cases.

3. Antibiotic treatment is terminated temporarily with the appearance of *Proteus*, *Bacillus aerogenes* or abundant colonies of *C. albicans* on sputum cultures.

4. Streptomycin with sulfonamides or chloromycetin may be employed for *Proteus* infections, and polymyxin B for *B. aerogenes*, unless these strains are also sensitive to streptomycin.

5. Prolonged treatment with penicillin, especially by aerosol, is safer than long-continued broad spectrum antibiotic therapy since patients may carry gram-negative bacteria on sputum cultures for years without infection and since instances of actual invasion by gram-negative bacteria may generally be controlled by adequate treatment, including the use of broad spectrum drugs. The latter agents, terramycin and aureomycin in 1.0 gm. daily dosage, are frequently of special value in the treatment of acute exacerbations of the infections that occur in the wake of the common cold or sore throat. Limited periods of five to six days of treatment are unquestionably useful in preventing pneumonitis or spread of the disease.

In interesting studies now in progress, therapeutic control of surgical infections has been achieved by a dosage of terramycin as low as 250 mg. twice daily; with the concomitant use of acidophilus organisms administered as tablets (LACTINEX), the develop-

ment of *Monilia* in stool cultures as well as the onset of diarrhea and loose stools have been avoided.[12] It would appear that unnecessarily high dosages of terramycin, and probably of aureomycin as well, have been responsible for adverse side effects that may perhaps be avoided if current studies on the effectiveness of the administration of smaller amounts is borne out.

Attempts have been made to facilitate bronchial drainage by liquefying bronchial secretions with the use of tryptar aerosol[34] and by an alkaline solution containing a detergent (Alevaire).[37] The latter mixture contains 1 per cent sodium bicarbonate which should be changed to potassium bicarbonate in cases with associated cardiac disease. The viscosity of sputum appears to be lessened by inhaling a watery aerosol; this can be most conveniently done, in our experience, by connecting a nebulizer to the plastic oxygen face tent.[22]

Although progress is being made in treatment of bronchiectatic infection by increasing bronchial drainage and by better control of antibiotic therapy, segmental resection of the more diseased parts of the lung appears to be justified in some cases even though residual bronchiectasis is present in other lobes. Removal of the most extensively involved areas may present a manageable problem even in multilobar bronchitis.

Summary

The medical management of bronchitis and bronchiectasis has been discussed in respect to the joint use of methods of increasing bronchial drainage and antibiotic therapy. Since prolonged treatment with the more powerful broad spectrum antibiotic drugs may be followed, especially in patients with debilitating chronic pulmonary disease, by invasion with previously nonpathogenic organisms, attempts to facilitate the arrest of chronic infection should utilize bronchial drainage as much as feasible, including newer mechanical methods of eliminating retained bronchial secretions.

Drainage of the bronchi may be aided by exsufflation with negative pressure, diaphragmatic respiration and allied breathing exercises, employment of the head-down position and effective administration of bronchodilator, viscosity-lowering aerosols, intermittent inspiratory positive pressure breathing and intermittent negative pressure breathing.

The preferred treatment of the common gram-positive bacterial infections is the use of penicillin or the newer drugs, magnamycin and ilotycin, whose spectrums are limited mainly to gram-positive bacteria, rather than the initial use of broad spectrum drugs. Aureomycin and terramycin are especially valuable in the treatment of infections with gram-positive bacteria, especially *K. pneumoniae*, for patients in whom improvement does not take place during penicillin treatment and for limited periods of five to seven days during acute exacerbations of infection. Chloromycetin may be required to treat infections due to organisms insensitive to other antibiotics, such as *Proteus*, some cases of *K. pneumoniae*, paracolon and *Staph. aureus*, despite the possible risk of blood dyscrasias. The administration of broad spectrum drugs such as terramycin, in much smaller dosages than those previously employed, offers the possibility of effective control of infection with fewer side effects than those which have accompanied the use of larger dosages.

Severe acute reactions have followed the intramuscular injection of the diethylaminoethyl ester of penicillin. In 4 cases observed in our clinic, the reaction appeared to be toxic rather than anaphylactic in origin and indicated that employment of the drug be investigated with dosages much smaller than those previously employed, namely, 200,000 units four times daily instead of single larger amounts.

References cited will be included in the author's reprints.

A Tuberculosis Program: Its Results and Implications

Pulmonary Tuberculosis *Can* Be Controlled

This requires:

Understanding the nature of the disease

Vigorous utilization of all resources and facilities

Early detection of cases

Adequate treatment

Effective rehabilitation

Periodic medical examination for the rest of life

Continuing research — clinical, pathological and social

Effective health education

These facts must be taught:

Pulmonary tuberculosis is a chronic disease, requiring prolonged treatment and supervision.

. . . . spread by the tuberculous, including patients or undetected cases who may appear perfectly well.

. . . . can be prevented by avoiding contact with the tuberculous.

Contacts require periodic roentgenologic examination.

Early detection and adequate treatment give the best chance for return to normal living.

These concepts are illustrated in the following charts, which deal primarily with the methods and results of a comprehensive clinical and research program that has led to the control of tuberculosis among employees of the Metropolitan Life Insurance Company. In addition, since 1909 the Company has been in the forefront of the campaign against tuberculosis through its continuous program of popular health education, its support of research, and many other activities. The downward trend of mortality from the disease, shown in one chart, reflects the impact of these efforts. A final chart summarizes the present status of the problem across the Nation. This exhibit was first presented at the annual meeting of the American Medical Association at Chicago in June 1952.

EARL C. BONNETT, M.D. • HAYNES H. FELLOWS, M.D. • DONALD B. ARMSTRONG, M.D.
LOUIS I. DUBLIN, Ph.D • HERBERT H. MARKS

METROPOLITAN LIFE INSURANCE COMPANY

THE PROGRAM FOR EMPLOYEES

EARLY DETECTION AND REDUCTION OF EXPOSURE TO INFECTION

Medical Examination of Employees

Annual — all employees

Special — employees with frequent or protracted illnesses, with symptoms, medical problems, or history of contact.

Since September 1, 1928 fluoroscopic examination of the chest has been a routine part of all annual examinations of employees and has been freely made at other times. This has been supplemented by roentgenograms, as indicated.

Pre-employment medical examination of applicants for jobs in the Company.

Since October 1, 1927 this examination has included routinely a fluoroscopic examination of the chest and since August 1, 1935 routinely a chest X-ray.

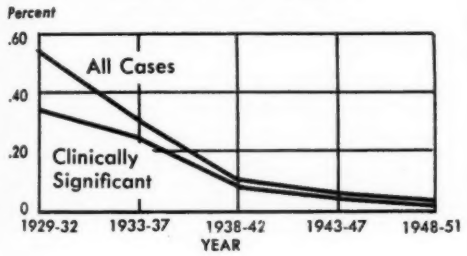
Re-check of applicants with active or suspicious tuberculous lesions; and referral to medical supervision for further diagnostic studies.

Results of Intensive Case-Finding Procedure

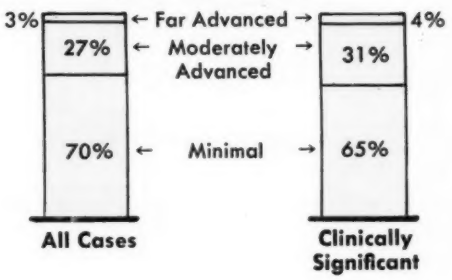
AMONG EMPLOYEES

Decreasing incidence of tuberculosis . . . . . yet high proportion discovered in minimal stage

Percent of New Cases of Tuberculosis Among Home Office Employees



Stage of Disease in New Cases



AMONG APPLICANTS FOR EMPLOYMENT

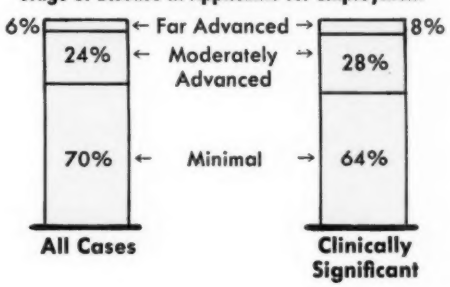
Many persons discovered with tuberculosis

Total discovered, 1928-1951..... 532
(0.9 percent of total examined)

Identified as active at the time..... 330
(0.5 percent of total examined)

These cases received the benefit of early diagnosis

Stage of Disease in Applicants for Employment



THE PROGRAM FOR EMPLOYEES

PROMPT AND THOROUGH TREATMENT

Principles

Treatment in a well staffed and well equipped sanatorium* until the patient is able to resume former or other suitable job full time. This care given without cost to the employee.

Lessening the economic burden as well as the mental and emotional impact of the disease on the patient and his family by providing cash disability benefits, and thus reducing to a minimum the number discharged against advice.

Type of Treatment at Company Sanatorium

Rest therapy — basic and sufficient alone to control the disease in most cases.

General medical and supportive measures

Surgery — as indicated

Rest Therapy as Practiced at Mt. McGregor

		Passage to successive phases guided
HORIZONTAL PHASE	Absolute bed rest	T.P.R. daily q. 4h.
SEMI-HORIZONTAL PHASE	Recliner or chair	X-ray film
SEMI-AMBULATORY PHASE	Toilet privileges	Sputum and/or gastrics
AMBULATORY PHASE . . .	Meals in main dining room	Symptomatology
EXERCISE PHASE (Walking) . .	Regulated increments up to unlimited	Physical examination Daily visit by physician 24 hour observation by nurses

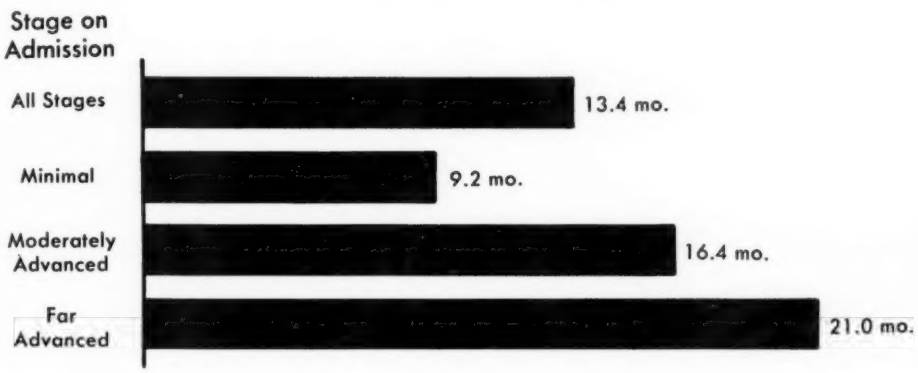
All patients except those on unlimited exercise required to rest in bed 2½ hours in the afternoon also, in bed or recliner, when not engaged in prescribed exercise.

*This refers to the Company Sanatorium at Mt. McGregor, New York, to which tuberculous employees were invited for treatment up to the time it closed. The John R. Hegeman Memorial Laboratory, where research in tuberculosis was conducted, was an integral part of the Sanatorium.

Results of Good Care and Treatment of Tuberculosis Under Favorable Circumstances

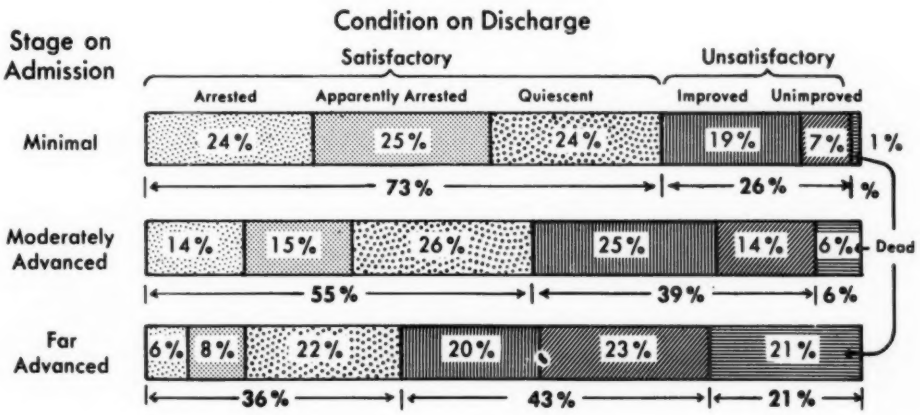
Patients more willing to stay under treatment long enough to receive maximum benefit

Median Length of Stay at Mt. McGregor



High proportion of patients in satisfactory condition at discharge, especially the minimal cases

Percent According to Condition on Discharge



First Admissions 1919-1945

THE PROGRAM FOR EMPLOYEES

SYSTEMATIC REHABILITATION AND FOLLOW-UP

Patients returned to former job unless it is unsuitable, with modification of work, as indicated.

Continuous medical supervision of ex-patients*

Regular and, as indicated, special medical check-up, including periodic roentgenogram and examination of sputum or gastric contents.

Periods of modified rest treatment, as indicated, at home or elsewhere, as prophylactic measure or following serious illness or surgery.

Prompt attention to signs or symptoms of reactivation, and resumption of treatment, if indicated.

*Complete program as carried out at Home Office

. . . . . .

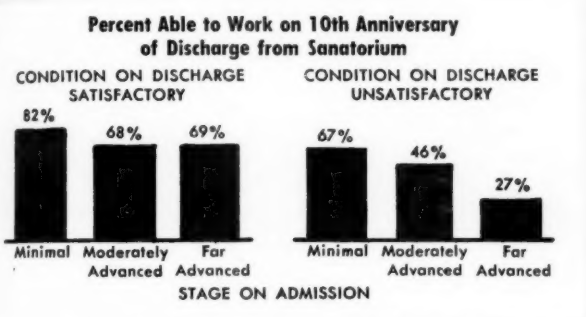
The tuberculous patient, when first told that he has the disease, is naturally fearful and depressed. On the basis of our experience, we can truthfully say to the person discovered to have minimal tuberculosis,

"Your chances of recovery are excellent. Your chances of dying are little, if any, greater than your fellow workers' who do not have tuberculosis. Your outlook for return to work is so good that you can look forward to it with confidence."

. . . . . .

Results of an integrated program of detection, treatment, and continuing supervision

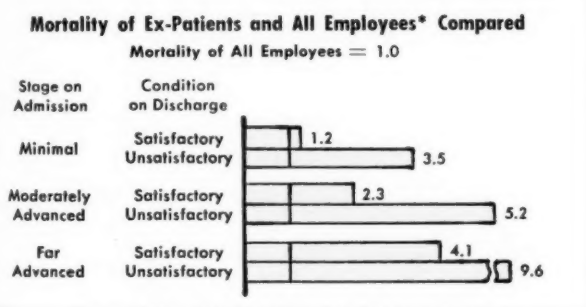
Ability to work restored



First Admissions 1919-1939, Discharged 1939 or Earlier

This is a unique record. All but a small fraction of these patients have been followed up successfully, and a great many of them had continuous and careful clinical observation. There is virtually no other post-sanatorium experience which provides so accurate information over so long a period.

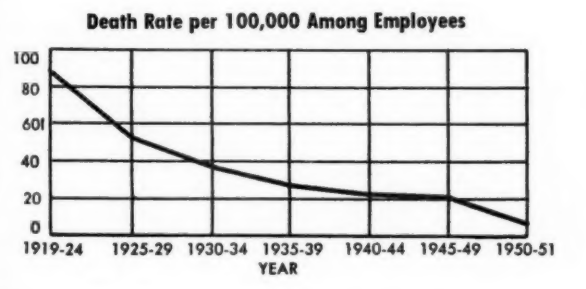
Subsequent mortality record of ex-patients is favorable



First Admissions 1919-1945 Traced to 1949 Anniversary

*Ratio of number of deaths observed in each group after discharge to number expected if ex-patients had same death rates, by sex and age, as all employees.

Death rate from tuberculosis among all employees sharply reduced

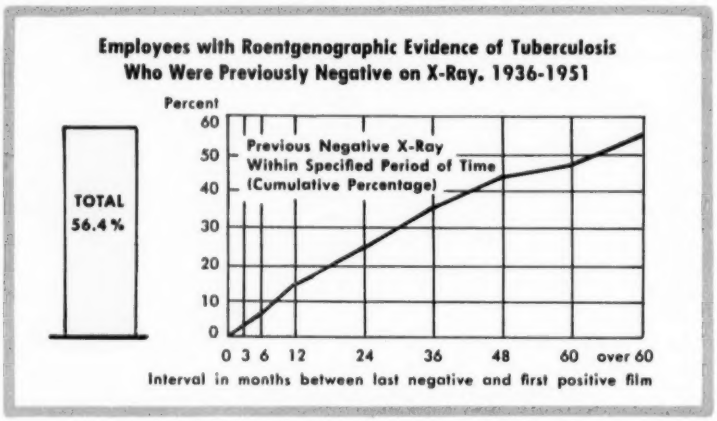


The death rate from tuberculosis among employees is now less than half that in the general population. The reduction in the rate also has been more rapid than in the general population, the decline since 1919-1924 being 90 percent and 79 percent respectively.

THE PROGRAM FOR EMPLOYEES

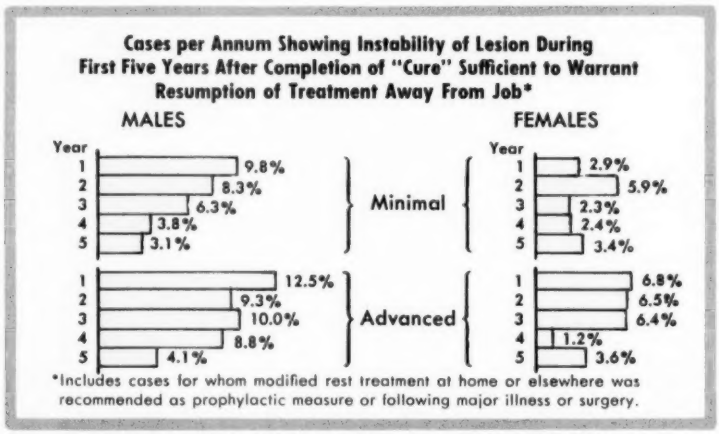
CLINICAL RESEARCH—MAJOR RESULTS OF PUBLIC HEALTH SIGNIFICANCE

A single negative X-ray film does not guarantee freedom from tuberculosis even for short periods



PERIODIC ROENTGENOLOGIC EXAMINATION VITAL FOR TUBERCULOSIS SUSPECTS AND CONTACTS • ADVISABLE FOR ALL ADULTS

Tuberculosis is a chronic disease and may take a long time to become stabilized

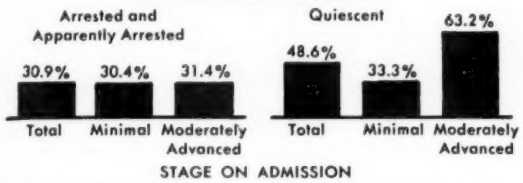


Based Upon First Admissions to Company Sanatorium, 1919-1944

THE TUBERCULOUS PATIENT NEEDS CONTINUING MEDICAL SUPERVISION EVEN AFTER APPARENT ARREST

The tuberculous patient always remains a potential spreader of the disease

Percent with tubercle bacilli in sputum or gastric contents following return to work after sanatorium treatment for tuberculosis

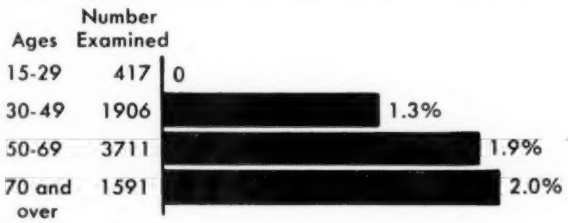


Based upon one or more positive findings. Average number of tests was 7.9. Experience during 1940-1943

THE PATIENT WITH "HEALED" TUBERCULOSIS MAY BE THE UNSUSPECTED SOURCE OF NEW CASES

Undetected open tuberculosis is much more frequent than is generally realized

Undiagnosed Tuberculous Cavitation Found at Autopsy

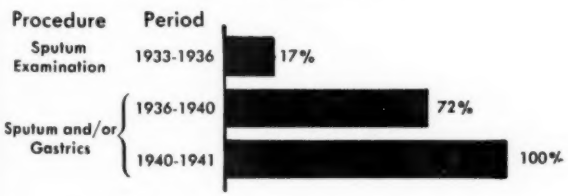


Based upon complete postmortem examinations at Bellevue Hospital, New York City, 1935-1945. The great majority were on white men over 40.

A MAJOR SOURCE OF NEW TUBERCULOUS CASES IS THE UNSUSPECTED AND UNDETECTED CASE WHO IS USUALLY A MAN OVER 40

Sputum examination is valuable even in the diagnosis of early tuberculosis

Percent with Positive Sputum or Gastrics among New Minimal Cases



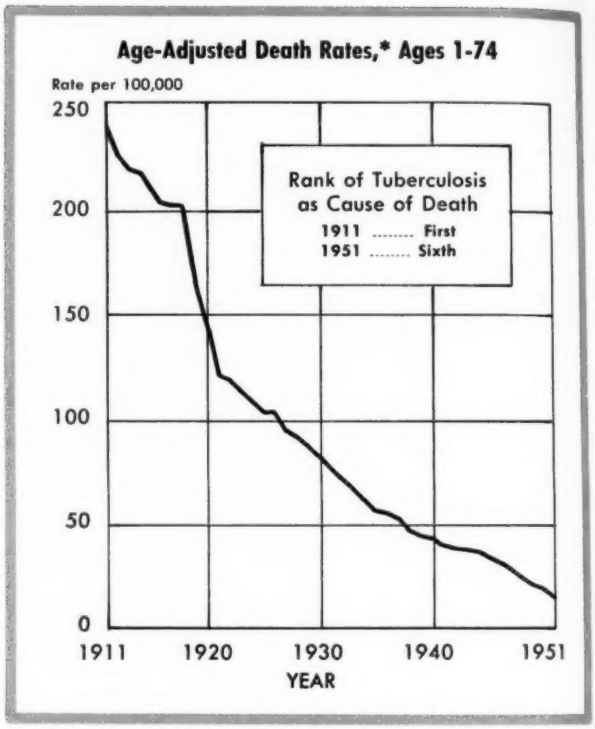
SPUTUM EXAMINATION IS A QUICK DIAGNOSTIC METHOD IN TUBERCULOSIS AVAILABLE TO ANY PHYSICIAN — MAKE YOUR OFFICE A TUBERCULOSIS CASE-FINDING STATION

The statistical illustrations are derived from studies, some not yet published, made at the Home Office and Sanatorium of the Company by E. M. Medlar, M.D., A. C. Reid, M.D., and M. G. Stephens. Some of these studies were supported by the John R. Hegeman Memorial Fund.

THE REDUCTION IN TUBERCULOSIS MORTALITY

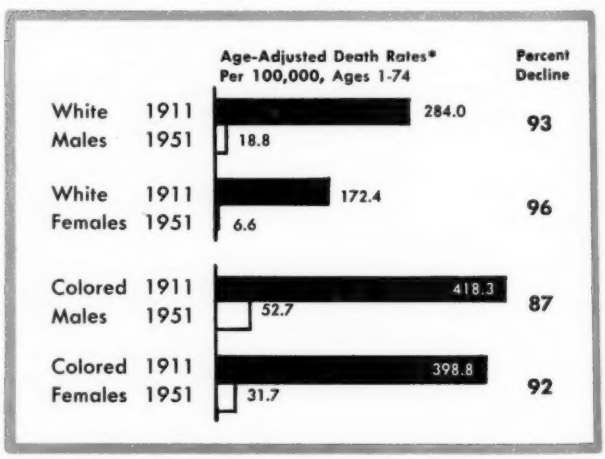
GENERAL TREND

The death rate from tuberculosis in insured wage earning families has been cut radically. It is now 94 per cent less than in 1911. Then the leading cause of death, tuberculosis is today sixth in rank.



COLOR AND SEX

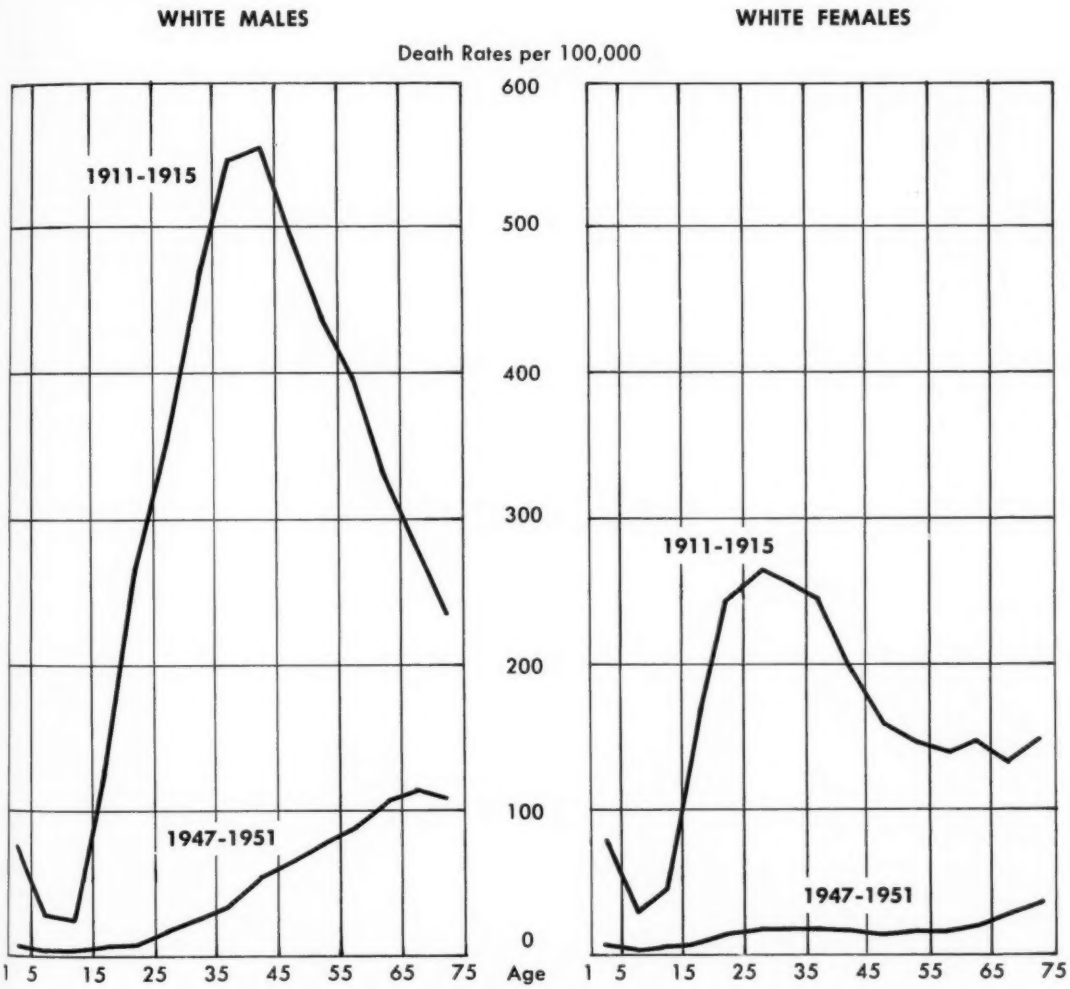
The reductions are extremely large in each sex and color group. The improvement has been greater in females than in males, and in white than in colored persons.



*Age-adjusted on basis of Standard Million of England and Wales 1901.

AGE AND SEX

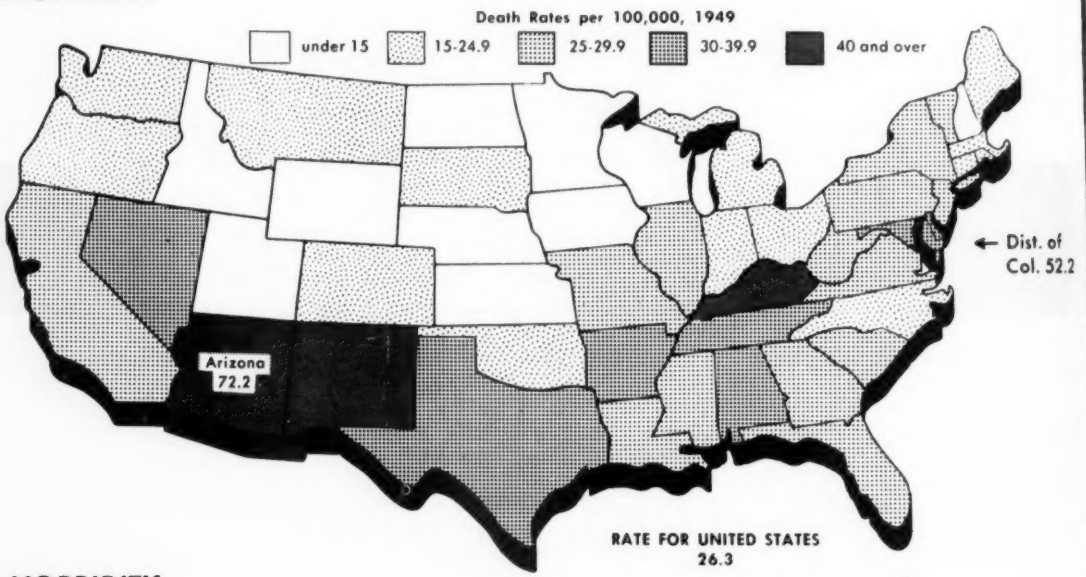
In every period of life tuberculosis death rates have been sharply reduced, but most at ages under 45. This has brought a marked change in the age curve of tuberculosis mortality. The peak of mortality has shifted to the later ages of life.



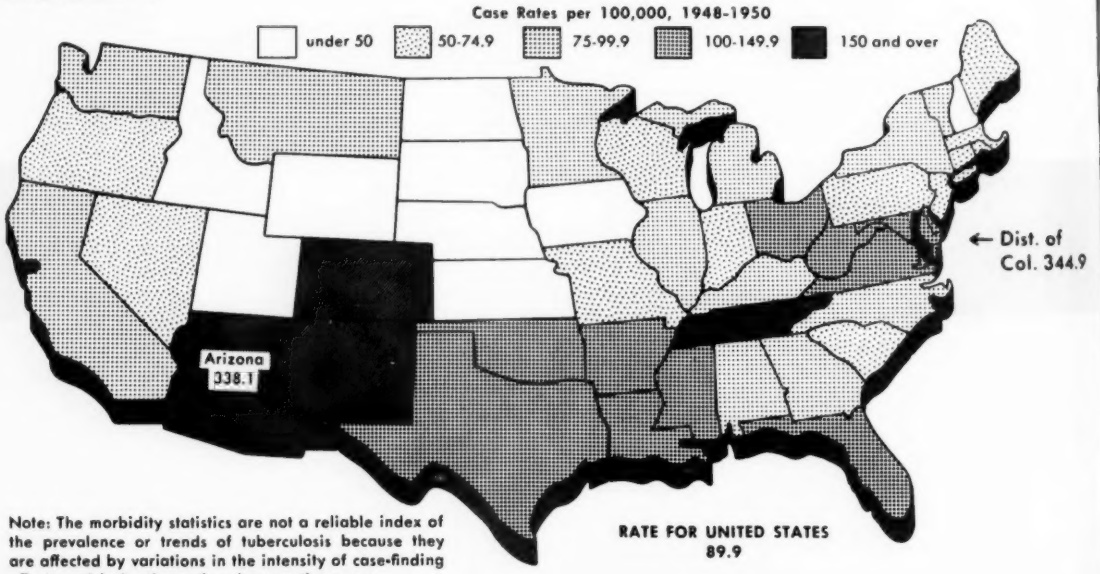
THE CURRENT TUBERCULOSIS SITUATION IN THE UNITED STATES

The tuberculosis problem in this country today is concentrated in families with low incomes and poor living conditions. This is indicated by the high death rate from the disease in the South and Southwest, due mainly to the high mortality among their nonwhite populations. The geographic pattern of morbidity is similar to that of mortality. The number of persons with tuberculosis has not changed radically. The total number of active cases is now estimated at 400,000 for the country as a whole, and new cases reported still exceed 100,000 annually.

MORTALITY



MORBIDITY



Note: The morbidity statistics are not a reliable index of the prevalence or trends of tuberculosis because they are affected by variations in the intensity of case-finding efforts and in local practices in reporting cases.

Pediatrics IN GENERAL PRACTICE

For general practitioners, who handle more than 85 per cent of all pediatric care, POSTGRADUATE MEDICINE *here presents authoritative, up-to-date summaries by specialists in pediatric problems.*

Feet and Shoes in Infancy and Childhood

LEE BIVINGS

Atlanta, Georgia

NEARLY all babies are born with good feet. Using a simple method of footprinting which I will describe later we have recorded the footprints of more than 5000 infants since 1932. The normal strong foot in infancy shows a well defined arch, narrow heel and broad forefoot. The normal relationship of heel to forefoot is easily shown by a line which passes through the center of the heel in its long axis and extends forward to cross the forefoot between the third to fifth toes.

LEE BIVINGS

This position of slight adduction is maintained until walking begins. Then the foot collapses by pronation of the heel and abduction of the forefoot on the heel. The foot then looks and is flat. This position holds until the leg muscles become accustomed to weight bearing and recovery of foot balance begins. This period of foot development, beginning about the end of the first year, lasts until the third, fourth or fifth year and is known as the "transition period."

Swartz and Heath[1] showed the human foot to be structurally weak because of two factors: (1) The line of weight comes down through the center of the tibia and is about 1.5 cm. medial to the center of the os calcis where it touches the ground, and (2) the rounded undersurface of the os calcis also tends to produce pronation. Therefore the infant first beginning to walk has great difficulty in maintaining normal foot balance.

The hingelike ankle joint does not allow much twisting of the heel on the leg but does allow some pronation, inevitably followed by abduction of the forefoot on the heel.

The muscles of importance are the anterior and posterior tibialis, which are involved chiefly in supination and maintenance of foot balance, and the flexor hallucis longus which maintains foot balance during the raising of the heel and assists in adducting and supinating the foot. The gastrocnemius and soleus do most of the work in propulsion but are not involved in the maintenance of foot balance except where shortening of the tendon occurs.

The most practical method to assist the child through the important transition period and to give him the best chance of good feet

in later life seems to be through the use of shoes which will help maintain foot balance while the muscles are becoming accustomed to weight bearing.

In a study of 1300 case histories,[2] I was able to show that of approximately 92 per cent of infants having strong feet before weight bearing, approximately 91 per cent had good feet by the fifth to the eighth year of life.

Shoe Requirements

The shoe should hold the foot in a position of slight adduction and supination which is the normal position of the foot in infancy before weight bearing begins.

The only important point of flexion in the shoe is under the ball of the foot; therefore the sole can and should be stiff enough to at least partially overcome the tendency of the foot to abduct. The heel should fit snugly with a heavy counter to prevent pronation. The fore part of the shoe should be of the combination-last type allowing plenty of width so that the great toe can be straight with the long axis of the foot. The fore of the shoe should be from once and a half to twice the width of the heel. If the shoe fits snugly around the ankle enough length can be allowed for several months of normal growth. The upper of the "high shoe," which all infants should wear, should not be high enough to interfere with free movement of the ankle—usually about 4 lace holes are enough.

High shoes seem of particular value in the first 12 to 18 months of shoe wearing (from 1 to 3 years of life) to help the foot overcome the natural tendency to pronate. And when pronation occurs the foot attempts to bridge across and break down the inner side of the shoe. Only a heavy shoe can withstand the pressure. A supinating wedge, 1/8 inch thick in small shoes to 3/16 inch in larger sizes, between the layers of the heel on the medial side of the shoe greatly helps the shoe to maintain normal foot balance. Such a wedge under the heel seems to be of greater value than the so-called arch support, because

Inner side heel wedge to straighten ankle from tipping to inside. Also used to spread knock knees
Outside toe wedge with inside heel wedge to stop toeing in.

FIGURE 2. Positions of wedges—the first to correct pronation and the second to correct toeing-in. Toe wedges should be slightly higher than heel wedges.

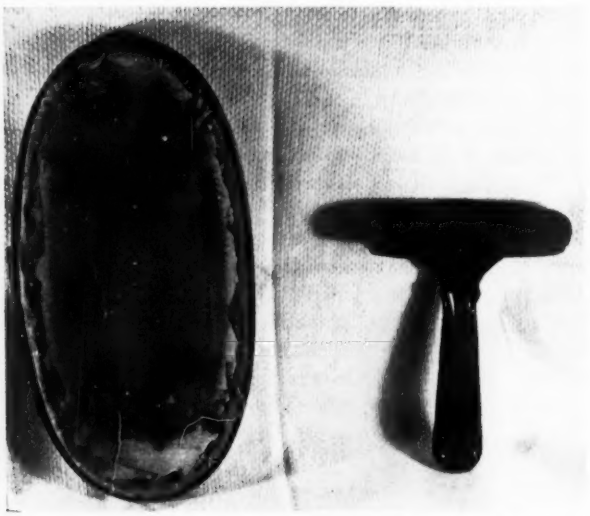


FIGURE 1. A simple footprinting device, made by stretching dental rubber dam over oval embroidery hoops.

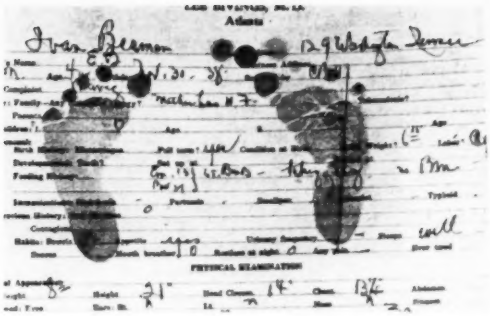
if pronation and abduction can be controlled the arch will take care of itself.

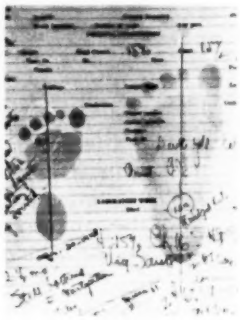
Heels should be fitted snugly; if the ankle area is fitted correctly the shoe can extend a thumb's width beyond the end of the great toe. The wearing time of shoes can be increased by cutting out the toe when the shoe becomes too short. Obviously shoes which are too short can harm growing feet and so can socks that are too short.

Many shoes which meet the requirements cited are commercially available. The soles should be fairly stiff; the heels should fit snugly with heavy counters inside the heels, and, for beginners, high shoes should be worn

for 1 or 2 years. Rubber heels often prevent prematurely misshapen heels and can be added at almost any age past infancy.

In the early years of this study I used swung-in shoes with Thomas heels for many children with pronated feet. After it became apparent that many were within the transition period it seemed unnecessary. A simple heel wedge in a strong, well-shaped shoe would usually suffice. For older children with prona-





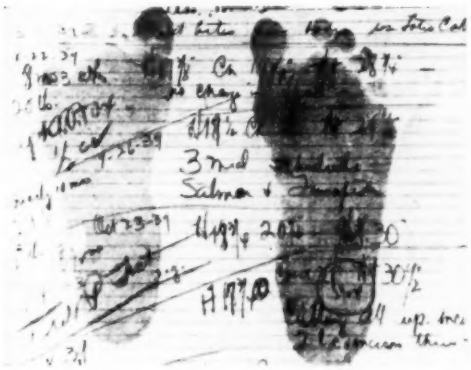


FIGURE 3a *(left)*. Normal infant foot showing footprint line and shape of good infant shoe. Second print shows pronation and abduction which occurs on weight bearing. b *(center)*. Pes cavus type foot showing collapse on walking but arch will be very high by 5 or 6 years of age. c *(right)*. Mild metatarsus varus before and after walking. No correction other than weight bearing. At 2½ years, feet are still in transition.

tion, such shoes are helpful in restoring normal foot balance. Several such shoes are commercially available.

About the beginning of the third year, well-built, low-cut shoes (laced) will usually maintain foot balance. Heel wedges (medial side) may be added when necessary.

Until after the transition period, barefeet, sneakers, rubber-soled shoes or sandals should not be allowed, except for brief periods, since none of these offers any foot support. If the feet are well balanced and strong it does not seem to matter. There is a type of sandal closely resembling an oxford in foot support which can be used in place of an oxford.

Footprinting

A method of footprinting is shown in Figure 1. An oval embroidery hoop covered with a dental rubber dam is inked on the under side with a felt-covered roller and stamp pad ink. The exact impression of the foot records on paper and no foot washing is necessary.

Undesirable Aids

Strollers—Walking is delayed by the use of strollers and there is a tendency for the infant to break down the inner long arch by pushing around in a stroller.

Foot exercises—These seem unnecessary for weak feet during the transition period; later they are helpful but of little value unless adequately supervised and maintained over a long period.

Arch supports—In the attainment of normally balanced feet I have never found it necessary to use arch supports, whether rigid or soft. I have felt that adduction and supination were more important and could be obtained more easily as previously described.

Associated Conditions

Knock knees are usually a result of pronation and usually disappear when pronation is

FIGURE 4. A well-designed baby shoe. Cutout shows heavy counter in the heel.

corrected with well-built, wedged shoes.

Pigeon or ding toes result from an attempt to shift weight to the outer side of the foot but if this is allowed to persist it will often become a fixed habit. First, correct pronation with wedged heels, then add higher wedges (1/8 to 3/16 inch) under the third to fifth toes. When toe wedges are used it is usually best also to add slightly lower wedges under the medial sides of the heels.

Pes cavus type—In my work with infants there are about 6 per cent who show very high arches even in infancy and this percentage increases to about 30 in later childhood. I have called these the "pes cavus type." When seen in infancy the condition is usually familial. The feet are and will be strong, although the resulting pronation sometimes is a source of leg pain and discomfort and requires heel wedging to correct. This condition is not true pes cavus which is an orthopedic condition and requires special care by a specialist.

Metatarsus varus—There is a mild form of adduction of the forefoot at the midfoot level which is a mild form of clubbing. I have observed more than 20 of these cases over a period of years and, if left alone, the pronation and abduction which take place at walking time will correct the problem.

Clubfeet—True clubfeet should have orthopedic care in very early infancy.

Effects of illness—Prolonged periods of debility may cause the feet to collapse. Restoration of foot balance can be attained in the same basic way as during the transition period of infancy.

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Intussusception in Infancy

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After the first few weeks of life, acute intussusception is the most frequent type of intestinal obstruction encountered during infancy. To me, intussusception has always been an intriguing subject, and I am ever amazed at some of the variations it may present. Sometimes it is present when you least expect it, and again when you are fairly sure of the diagnosis it is absent. Why it is not suspected in the early hours of the disease and why immediate steps are not taken to prove or disprove it is difficult to understand. In my opinion this condition represents the gravest incident that can occur within the abdomen of the healthy, well nourished baby, and it behooves every physician to substantiate or rule out the entity once suspicion is aroused. Too frequently the diagnosis is made

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late or not at all, and what should have been a comparatively simple cure is converted into a catastrophe.

In many instances the diagnosis can be made by telephone if you will just take time to listen to the mother tell her story. Any infant who has passed blood and mucus by bowel must be seen by the physician within the hour, and to give the mother an assuring answer or prescribe a palliative procedure over the telephone is pure neglect and abuse of our obligations and responsibilities.

The infant, particularly one less than a year old, who is stricken with intermittent abdominal pain should be suspected of having intussusception and be examined at once. The onset may be violent, striking like a "bolt out of the blue"; it may be of moderate severity or even mild in nature. If the onset is violent, you are in all probability dealing with a tight intussusception and the severity of the attack will produce shock. If the pain is moderate to mild, no shock will be present. I definitely feel that our textbooks should be revised regarding shock in intussusception in infancy. Fully 50 per cent of babies with intussusception do not present shock. The wee one may vomit with the first attacks of pain, but vomiting is not an early symptom in acute intussusception. Numerous investigators who did some of the early studies on intussusception felt that assumption of the knee-chest position during an attack of pain was almost pathognomonic for enterocolic intussusception. A normal bowel movement early in the disease should not deceive you; it is merely an evacuation of the rectum resulting from violent peristalsis. Within a few hours blood and mucus will be passed by bowel, the so-called "red-current jelly" stool; rarely does this appear until six hours after the onset.

With such a history, abdominal examination will reveal an empty right lower quadrant and usually a palpable mass. If any question arises regarding the mass, or the baby is difficult to examine, administer chloral hydrate by rectum; then, with the baby asleep, thoroughly palpate the abdomen. This procedure will usually elicit the abdominal tumor. Given the triad of (1) intermittent abdominal pain, (2) blood and mucus by bowel and (3) palpable abdominal tumor, the diagnosis of intussusception is certain.

If there is still doubt regarding the diagnosis, take a flat plate of the abdomen. From the flat plate alone a competent roentgenologist can usually outline the head of the intussusceptum and confirm your impressions. I think that at this stage a barium enema may be administered by experienced and skillful hands. The argument continues as to whether a barium enema is justified for diagnostic or therapeutic purposes. Recent publications from good clinics would lead us to believe that about 85 per cent of acute intussusceptions in infancy can be successfully reduced by barium enema. I firmly believe that many intussusceptions can be reduced by barium enema, when seen early, perhaps in the first 4 to 12 hours. Perhaps 50 per cent of the early intussusceptions that we see are reduced in this manner. On the other hand, given a late intussusception or a baby in severe shock, I believe it is folly to attempt reduction by means of a barium enema. This type of intussusception presents, beyond doubt, a surgical problem.

In years past when one thought of intussusception he immediately thought of surgery. Now I presume it is heresy for a pediatric surgeon to talk about the nonsurgical management of acute intussusception in the infant. On the contrary, over the years I have become convinced that about 50 per cent of early intussusceptions can be treated safely in this manner. As a result the management of early acute intussusception becomes as much a problem for the pediatrist and roentgenologist as for the surgeon. All of us have seen intussusceptions within the first few hours which have spontaneously reduced.

The small filling defect frequently seen in the region of the ileocecal valve, without barium passing into the terminal ileum, should not lead to immediate surgical intervention. Return the infant to bed, and if he is symptom free, prepare the enema in 4 to 6 hours. Our experience has been that the filling defect disappears and the terminal ileum can be readily visualized. If the baby continues to

have intermittent abdominal pain, surgical intervention must be instituted without further delay.

Certain pitfalls and dangers must always be kept in mind when reducing an intussusception with a barium enema. I cannot agree that it is quite as easy or safe as some would lead us to believe. The dangers of such a procedure are (1) incomplete reduction, (2) injury to the appendix and its blood supply, (3) overlooking an enteric intussusception initiated by a Meckel's diverticulum and (4) rupture of the intestine. I think, however, that with acute observation and repeated examinations you can avoid these dangers and keep the infant out of trouble.

I have seen more intussusceptions recur after surgical reduction than following reduction with a barium enema. One must always bear in mind that surgical interference necessarily carries a certain morbidity and that surgery must not be resorted to unless, as I have outlined, it is definitely indicated.

Preoperative preparation must be done with the utmost care. A thorough gastric lavage must be done and an indwelling gastric catheter put in place before going to surgery. Shock, if present, must be treated, and an intravenous catheter must be in place and functioning properly. A glucose drip must be flowing, with plasma connected and blood readily available. Plasma will usually suffice to combat the operative shock, but if much blood has been lost whole blood must be given. With the extremities wrapped in sheet wadding to conserve body heat, and a room temperature of 85° F., the baby is ready for the operation.

With the baby placed in slight Trendelenburg position and under ether anesthesia, by an open mask, a long right rectus incision will give the proper exposure for at least 90 per cent of all intussusceptions, which are enterocolic in type. Upon opening the peritoneal cavity, free straw-colored fluid is encountered. Locate the mass at once and bring it into the operative field under direct vision. I think it unwise to attempt blind reduction for fear of rupturing the bowel and losing the baby as a result of peritonitis.

Once the mass is in the hands of the operator and the extent of the intussusception is visualized, reduction is started. With one hand on the entering bowel and the other at the head of the intussusceptum, reduction is executed by pressure on the head and the most gentle traction on the entering bowel. If the neck is extremely tight, a hemostat may be inserted alongside the intussusceptum and gently spread to dilate the neck. Once the reduction starts it will progress smoothly until the head reaches the neck; then if the remaining mass is gently squeezed with a warm pack, satisfactory reduction can be completed. If the intussusception started with a dimple on the lateral wall of the cecum, completely evert the dimple. Inspect the junction of terminal ileum and cecum closely to make sure that reduction is complete. Search for a Meckel's diverticulum which may have started the intussusception, and if found, remove it. If the appendix has been caught in the intussusception, remove it. Perhaps more frequently than we realize, appendiceal pathology may initiate an intussusception. The thickened leather-like feeling of the terminal ileum is merely due to hypertrophied lymphoid tissue; do not open the ileum or cecum and expect to find polyps. Terminate your procedure as quickly as possible, with meticulous closure of the abdominal wall. Carry the baby back to his crib with head down, place him in the Trendelenburg position; tie all 4 extremities.

If resection has been necessary or if the appendix has been removed, give antibiotics for 72 to 96 hours. We employ rather heavy sedation for 48 hours to prevent crying and straining. Hypoclysis is necessary for the first 24 hours; water is given by mouth thereafter. Feedings may be started on the third postoperative day if tolerated. Administer vitamin C for first week postoperatively to assure better wound healing. Keep the baby flat on his back for 10 days to prevent possible evisceration.

In closing may I again emphasize that careful history, early examination and diagnosis, followed by immediate reduction, will markedly reduce the mortality rate of acute intussusception in infancy.

Editorials

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TRAINING CENTERS IN THERAPEUTIC RADIOLOGY

THE benefits to be derived from an actual division of the practices of diagnostic and therapeutic radiology have been eloquently presented[1,2] and the virtues of and the necessity for a general practice of radiology have also been well defended. However, the best present means leading to a higher standard of practice of therapeutic radiology are not agreed upon. To call for a division of large departments of radiology into departments of radiodiagnosis and of radiotherapy is to ask for delivery without gestation, for, at the present time, even cancer hospitals with a high sense of responsibility for the lives of their patients must do without radiotherapists for lack of well qualified aspirants to the positions. To divide departments of radiology and to entrust radiotherapy to self-trained neophytes may prove to be the hardest blow yet given to therapeutic radiology. To stiffen the specialty board examinations would result only in penalizing young students for a poor quality of training for which they are not responsible; and to require special certification by a radiotherapy board of examiners offers no better solution.

It matters little that radiotherapy will continue to be administered chiefly by general radiologists; but it is highly important that new aspirants be given commensurate training in both radiodiagnosis and radiotherapy, and that they be made conscious of their responsibility to the cancer patient. Adequate training in radiotherapy implies instruction in the physics of radiations, enriched by versatile exercise in its everyday utilization. The aim should be to make the student well aware of the possibilities and limitations of the means in his command. As Stead has said: "It is surprising how far it is possible to go on giving the learner a real insight into physical principles without covering the blackboard with a mass of symbols and equations."[3] A thorough knowledge of radiobiology is also required and ideally this should be accompanied by some firsthand experience in experimental work. Whereas there is more to radiotherapy than the treatment of tumors, the therapeutic radiologist requires primarily a vast knowledge of pathology of tumors, without which his other abilities take a lesser significance. By knowledge of pathology of tumors is not implied a mere pretense of microscopic recognition of specific morphology but also a thorough acquaintance with the natural history of tumors (rate of growth, modes of spread, radiophysiologic response) and of their dynamic character, which is acquired by prolonged contact with tumor patients. Gross pathology as observed in the autopsy room and the elements of microscopic diagnosis are a useful complement.

But, above all other essentials, the radiotherapist must have a wide *versatile clinical training*. Mayneord, the outstanding figure among British radiation physicists, has said: "No one with experience of the practice of

This editorial is a summation of remarks presented before the nineteenth Annual Conference of Teachers of Radiology at Chicago, February 1952.

radiotherapy would deny that fundamentally the radiotherapist is a clinician with all that that implies. His technical training is obviously secondary to his medical competence."[1] Inability to meet, examine and evaluate his patients makes many a radiologist appear as a disinterested technician, in the eyes of those patients. The training should be so organized as to facilitate constant interchange with cancer surgeons and tumor pathologists. Adequate facilities should be available for the examination of patients, new and old, and complete clinical histories should be available. Follow-up examination of patients should be especially emphasized and these appraisals should be attended by surgeons, pathologists and other specialists concerned, because they offer a true opportunity for lively interchange and correctional education. It should be obvious that the necessary instruction cannot be given in six months.

Radiologists with little interest in radiotherapy have permitted or stimulated the dissemination of the practice of radium therapy. Because of this, it is easy to judge the quality of training given to a resident by his deficiencies in radium therapy. Although the role of curietherapy has decreased in the field of therapeutic radiology, a radiotherapist must be given an opportunity in the personal handling of radium. Also the duties of a therapeutic radiologist today require training in handling radioactive isotopes.

The indifference which comes with inadequate preparation is contagious. If the young radiologist is to be raised to the realization of his duties in therapeutic radiology, his training must be directed by men who consider radiotherapy as their vocation, not as their hobby. Young radiologists cannot be inspired to their task in radiotherapy by being compelled to deal with incurables and with failures, while their admired chief enjoys himself behind the light boxes. And while the master heroically shortens his own life expectancy by his endeavors to discover early cases of cancer of the stomach with some chance of cure, his pupils should not be permitted to become indifferent to the plight of patients with cancer of the cervix who could survive, were their fate not left to routine and apathy.

The solution to this large problem lies in the future. It depends primarily on our ability to develop dependable *centers of training* in therapeutic radiology. We need first to train a good number of therapeutic radiologists with academic inclinations, capable of undertaking, in leading educational centers, the responsibilities of large departments of therapeutic radiology; this includes the adequate therapeutic training of general radiologists. In this manner a high quality of radiotherapy instruction will constantly be available to the young trainee, and this will stimulate the desire to receive adequate training. It also will make the aspirant more conscious of his high responsibilities in undertaking the practice of radiotherapy. It will be timely then to raise the minimum requirements of training set by the specialty board. And as increasingly capable men secure better results in the lifesaving aspects of radiotherapy, it will encourage others to choose to be trained exclusively in the highly exacting, strictly therapeutic aspects of radiology.

J. A. DEL REGATO

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FIDDLING AND DOODLING

As one travels about the world nowadays, one comes upon evidence that people everywhere are finding the tension of modern living a little strenuous and are seeking outlets and substitutes to divert this tension into either useful or harmless diversions. In Greece, even on the newsstands, strands of amber beads are sold to busy businessmen and disturbed housewives as a toy for diverting mental tension. Men who give up smoking carry such strands of amber beads behind them and play with them while walking about or even while sitting in the evening in social gatherings. One finds these strands of beads everywhere. They are for sale in imitation amber at low prices

and in real amber at exceedingly high prices. Even in New York City, so-called sterling silver fidget sticks are being sold for the use of nervous executives. They are shaped like spoons and are to be played with as a substitute for smoking, pencil chewing, knuckle cracking and doodling. Doodling pads have been for sale for some years. Certainly it might be worth while for some of our more practical psychologists or psychiatrists to give serious thought to this question of substitutes and tension relievers with a view to finding useful methods for aiding those who require something with which to either play or work as a means of relieving nervous tension.

MORRIS FISHBEIN

LABORATORY DETERMINATIONS OF BLOOD CONSTITUENTS

BIOCHEMISTRY has assumed an increasing importance in the diagnosis of disease. Obviously the results of biochemical tests are significant only when the determinations are made by competent clinical pathologists or biochemists and when their accuracy is unquestioned. If the range of normal values is exceedingly narrow, many people will have values which fall outside the normal. For that reason leaders in chemical pathology in Great Britain have recommended that any determination should be accompanied by a statement of the percentage of normals which it includes.

In the London *Lancet* (March 7, 1953) Drs. I. D. P. Wootton, Lecturer in Chemical Pathology in the University of London, and E. J. King, Professor of Chemical Pathology in the University of London, have discussed the interhospital differences that prevail as far as normal values for blood constituents are concerned. They submitted a series of specimens to a number of laboratories in order to find the agreements and the differences that occurred. The result of this experiment was evidence that a large disagreement exists between different laboratories due to obscure causes. They feel that a regular check on laboratory performance would obviously be useful and suggest, in fact, that this be done either in individual laboratories or on an area or national basis. As a result of their study they have invited laboratories in Great Britain to participate in further study and to communicate with them.

A similar survey was attempted in Pennsylvania about 5 years ago, using some 200 hospitals supervised by clinical pathologists. Similar results were obtained and at times individual laboratories found even wider variations than might be found between different laboratories.

Among suggestions that have been made as to how the difficulty could be overcome are the following: (1) the establishment of a special bureau of standards for laboratories of clinical pathology; (2) the establishment by hospitals of standards for all determinations; (3) the selection of a supervising biochemist from a large hospital in an area to supervise and educate clinical pathologists and biochemists in the smaller hospitals in his own area and, finally, (4) graduate study made available to biochemical technicians to improve working technics and to learn new technics.

Since modern medicine leans more and more on the laboratory, the importance of accurate standardization of methods and results is obvious.

MORRIS FISHBEIN

IMPROPER BEHAVIOR AND THE DOCTOR'S REPUTATION

IN Great Britain the Medical Disciplinary Committee of the General Medical Council is the body authorized to hear complaints on physicians and their actions. Sometimes cases are heard "in camera" and in other instances charges are heard openly. Most of the cases are exceedingly well reported in the *British Medical Journal;* they offer material of interest to every physician and a good deal of information as to the hazards that beset doctors in their practice.

Recently the committee heard a charge against a physician who, it was said, had been guilty of infamous conduct in a professional respect.[1] It was charged that, on numerous

occasions beginning in April 1949 and continuing to December 1951, he had behaved improperly to and committed adultery with a young woman whom he served in a professional relationship. Incidentally no charge had been brought before the civil courts, charging the doctor with committing a felony. The young woman testified that she consulted the doctor about her daughter, aged 5, and later became a patient herself. At about the fifth visit, she said, the doctor recommended hypnosis and she was hypnotized on four occasions. On each occasion, except the first, she charged the doctor behaved improperly to her. After having been hypnotized four times, she was accustomed to meeting him and going out with him in his car, which she said she did practically every Friday evening for two and a half years. She said they went to a spot near a disused air field seven miles from Leeds and on each occasion intimacy took place in the back of the car. She was never a willing party, she charged, and though not under hypnosis insisted that she was under the doctor's influence and that he more or less made her continue the association. An incidental light touch is the statement that on each occasion when they got to the place he would cover the British Medical Association badge on his car radiator. Finally, two years later she said she broke off the association with the doctor when she learned that his wife was pregnant. Her fiance testified that she appeared unhappy and depressed and that he had seen the doctor who admitted that he had given her hypnotic treatment. The doctor further is said to have reported to the fiance that he was pleased that marriage was contemplated and that it would be a good thing for her.

On the opposite side a night sister at the hospital testified that at an hour when it had been alleged that the doctor was with the lady in his car seven miles away he was, in fact, at the hospital attending a patient who had had an operation the previous day. Another witness said that at this time the doctor's car was in his garage for repair. The doctor himself said that he had a national health list of 2100 and many private patients, that he lived happily with his wife and two children, strongly denied he ever hypnotized the young woman or had any sexual relations with her. He had on occasion practiced hypnotism for anxiety neurosis but most of his patients knew all about it. He said that he had not taken the young lady out every Friday evening, that he had been occupied in social occasions at home except for emergency calls. In a word, he said the allegations in the complaint were completely malicious and a lie. His wife also testified to a happy marriage relationship. She too said he was home on Friday evenings except for an occasional brief call on some patient. Finally, it was brought out that the young woman had never made any complaints about the doctor's behavior to anyone close to her, including her mother. The committee then dismissed the charges.

This case is typical of many which occur and in which inquiring bodies have to determine whether or not the charges are made in good faith or represent some mental disturbance or a malicious action. The British doctor involved in the present case was believed by the council which was convinced that the charges had not been true. Nevertheless the publication *in toto* of such cases raises a mass of gossip and does an injury to a reputation which is difficult to overcome.

In the *British Journal of Medical Hypnotism* a discussion appears by George Newbold[2] entitled "Some medicolegal problems and the hypnotism act." Dr. Newbold emphasizes the frequency with which patients under hypnosis make false accusations against their physicians and recommends that a third party be present either openly or concealed when a male physician is using hypnosis with a female patient. However, he insists also on absolute secrecy and on refusal of the physician to disclose to the courts without the proper consent of a patient any information which may develop under such circumstances.

MORRIS FISHBEIN

[1]Report of General Medical Council, Medical Disciplinary Committee. Supplement to Brit. M. J., June 6, 1953, p. 263.

[2]Newbold, George: Some medicolegal problems and the hypnotism act. Brit. J. M. Hypnotism 4:12 (Spring) 1953.

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THE MEDICAL Bookman

► DERMATOLOGY: ESSENTIALS OF DIAGNOSIS AND TREATMENT

By Marion B. Sulzberger, M.D., Professor and Chairman, Department of Dermatology and Syphilology, New York University, and Jack Wolf, M.D., Associate Professor of Clinical Dermatology and Syphilology, New York University. Ed. 4. 592 pages with 65 figures. 1952, The Year Book Publishers, Inc., Chicago. $10.00.

The authors carefully explain that this book is an improvement and a development of their original text, "Dermatologic Therapy," which has enjoyed 3 editions. The present text contains much more material and represents a broader coverage of the subject.

The book begins with general dermatologic management and incorporates principles of medication, including careful detailed instructions on types of medication to use, proper indications for them and correct methods of application. It has always been important and common knowledge to the dermatologist that patients require careful instruction as to the proper use of medication. It is therein that the success of various medications lies. In addition to topical medication, we find principles of other forms of medication which include physical agents, such as x-ray, ultraviolet, heat and electrolysis.

The various dermatoses are taken up under such important headings as the eczematous dermatoses, urticaria, dermatoses due to fungi, pyodermas, etc. In a separate chapter, atopic dermatitis is explained in a manner that can be clearly understood by both the general practitioner and the student of dermatology. There are also discussions of common tumors of the skin and special miscellaneous dermatoses, such as lupus erythematosus, drug eruptions, etc., as well as the diagnosis and treatment of syphilis. Important among the many good illustrations are those concerned with the method and means of applying therapeutic agents. Also there are, to a lesser degree, illustrations concerned with diagnosis, although one must bear in mind that the diagnostic features are not emphasized in a text of this sort.

The authors further point out that this text is intended primarily as an introduction for the medical student, the graduate and postgraduate student in dermatology and for the practicing nondermatologist. Dermatologic specialists, too, will find it contains much useful information. A list of references for special reading is appended.

I. F.

► TREATMENT OF MENTAL DISORDER

By Leo Alexander, M.D., Director, the Neurobiological Unit, Division of Psychiatric Research, Boston State Hospital. 507 pages with 143 figures. 1953, W. B. Saunders Company, Philadelphia & London. $8.50.

The nonpsychiatrist is interested primarily in persons with neuroses and with the minor types of psychosis in which the person is well behaved and never has to be locked up.

There is much in this excellent book which can be of interest to the nonpsychiatrist. Most of the text is devoted to the best discussions of electroshock treatment which we have seen. Today psychiatrists are much at variance in their ideas of electroshock; some are even hostile to it. Some tend to make a big operation out of it, giving anesthesia and perhaps curare, while others give the treatment in their offices without much help and without any drugs. Some use an old type of apparatus which uses perhaps 450 milliamperes while others employ an apparatus which uses perhaps 10 milliamperes.

(Continued on page A-42)

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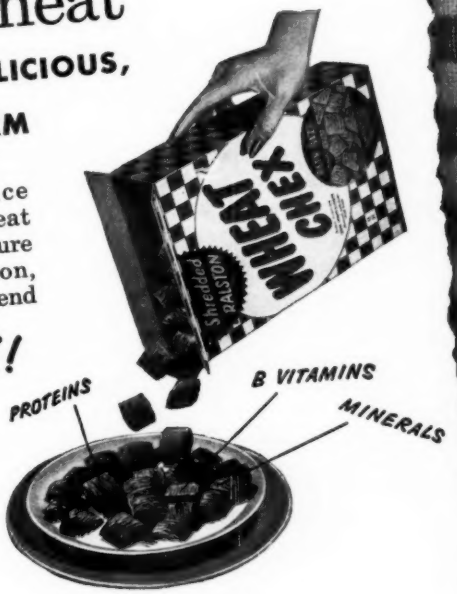

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Certainly it would seem that all psychiatrists should try to decide on the best procedure and the best type of apparatus and current. It is to be hoped that other psychiatrists will study and discuss the problem as thoroughly as Dr. Alexander has done.

The book is a bit technical for most physicians, but there are many parts of it which will interest them, which will be understandable to them, and which can help them in their practice. There are interesting and valuable chapters on the general principles of psychotherapy, on the complications of physical treatment, and on the brain damage which can be produced by physical therapy. Brain damage can take place during an insulin shock, and efforts should be made to avoid it.

There is also a chapter on the indications for physical treatment, based on psychiatric and psychosomatic clinical findings; and there is much detailed information on giving treatments. Good bibliographies follow each chapter. The book would appear to be a must for all psychiatrists who use electroshock therapy.

W. C. A.

► GYNECOLOGY: DISEASES AND MINOR SURGERY

Vol. 1. Compiled and edited by Robert J. Lowrie, M.D., Associate Clinical Professor of Obstetrics and Gynecology, New York University College of Medicine. 806 pages with 775 illustrations. 1952, Charles C Thomas, Springfield, Illinois. $22.50.

Forty-three physicians, mostly gynecologists of well established reputation, have contributed to this textbook. The first volume is devoted to medical gynecology and includes a chapter on minor surgical procedures; it apparently is to be followed by a second volume on major gynecologic surgery.

The editor has endeavored to gather opinions from many different sources and to compile this information into a text by having the contributing authors, wherever feasible, follow a uniform plan of presentation. The reader will find a fairly complete exposition of the basic information required to diagnose and treat gynecologic conditions in an intelligent manner, except for descriptions of major operative technics. Although the information is complete, it is not always uniform and is sometimes controversial. This is to be expected in a text assembled from many sources, including two foreign countries (England and Canada). However, the frequency of redundant or inaccurate and, in some instances, obsolete discussions testifies to the need for careful editing by someone who isn't afraid to step on the literary toes of the contributing authors.

The chapter on physical therapy is one of the longest in the book. It contains considerable material of a general nature and finds application for this branch of medicine in a much broader manner than is commonly accepted as good practice. The reviewer questions whether this chapter should have been included in its entirety. Finally, the chapter on minor surgery, although sharing the title of the text, occupies only 24 pages, in contrast to the 42 pages occupied by the chapter on physical therapy.

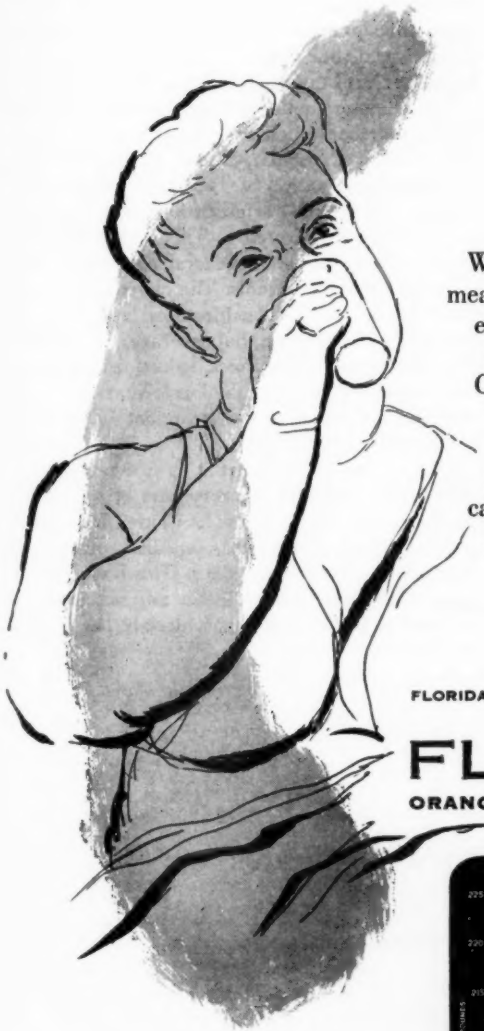
On the positive side of the ledger are some excellent chapters by such authorities as Meigs, Rutherford, Kennedy, Schauffler, Pemberton, Corscaden, Scott, Emge and Kleegman. The chapters on anatomy, physiology and normal and abnormal development are excellent. The illustrations are clearly portrayed and well reproduced. Most of the chapters have complete bibliographies, and the entire text has a complete subject and author index.

The criticism expressed is not intended as a blanket indictment of what must have been a monumental task for the editor. Certainly the book contains much of academic and practical clinical value for the student and practitioner of gynecology. It seems strange, however, to have in one text the scientifically presented material of international authorities coupled with material in other chapters based on somewhat less than scientific fact.

Future revisions of this first edition should be directed toward greater uniformity in the quality and the quantity of the material presented. The text is recommended for discerning readers who will overlook some of the defects and utilize the more digestible material, which fortunately comprises the bulk of the book.

M. B. S.

(Continued on page A-44)



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* *Postgrad. Med. 9:106, 1951.*

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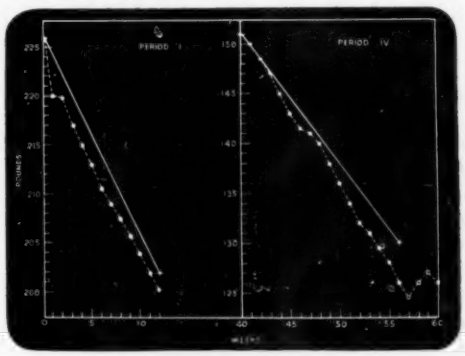

CHART OF WEIGHT LOSS
BROKEN LINE—OBSERVED LOSS • SOLID LINE—PREDICTED LOSS

► THE PHARYNX: BASIC ASPECTS AND CLINICAL PROBLEMS

Edited by Abraham R. Hollender, M.D., Professor of Otolaryngology, Emeritus, University of Illinois College of Medicine, Chicago. 560 pages with 31 figures. 1953, The Year Book Publishers, Inc., Chicago. $15.00.

A group of collaborators from clinical and basic sciences have written this text, limited to an anatomic region. Most of the writers are otolaryngologists but among other fields represented are: allergy, psychiatry, medicine, pathology and radiology. Most of the chapters are satisfactory compilations of known material which has been covered well in other textbooks.

The chapter on radiology seems to be very complete. The presentation of the x-ray films is done in *positive* rather than in *negative*. Usually when a clinician examines x-ray films he examines the negative. Therefore it is slightly less advantageous to view films in a textbook which are opposite to those viewed in examination of patients. This is not a serious objection but in the reviewer's mind does detract somewhat from the chapter on radiology. The chapter entitled "Oral and Pharyngeal Manifestations of Diseases of the Blood," by Dr. H. James Hara, is a particularly good one and brings together in one place a great deal of information not usually available to the otolaryngologist in his ordinary sources of literature. This chapter would be of value to a general practitioner dealing with a patient who had bleeding from the nose or mouth, or oral manifestations of blood diseases.

The chapter entitled "Acute Sore Throat" brings together much information but contributes nothing particularly new. The chapter on "Surgical Elimination of Tonsils and Adenoids" begins with a list of indications for tonsillectomy and adenoidectomy. This list of indications certainly is not in general agreement with more conservative schools. The chapter on "Surgical Technique of Tonsillectomy and Adenoidectomy" is adequate but average in quality. The chapter on "Deep Neck Infections" is good and contains a great deal of information which would be of value in the treatment of patients having this condition.

In a review of this sort it is not possible to catalog one's opinion of every part of a book of this type. And like most books written by a good many different authors this one has some good chapters and some poor ones. The book would probably be most useful as an adjunct text for the general practitioner, who already has a general text on otolaryngology.

R. E. P.

(Continued on page A-46)

The Medical Muse

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The whirs and rasps and wheezy gasps
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The stethoscope's a nice device
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Of sundry leaks and burps and squeaks
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For private hearing merely.
The patient can't hear ping or pant,
Although he'd love to, dearly.

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Things are often said quite biting
About a doctor's way of writing—
The hieroglyphic tracks of hen
That burgeon underneath his pen,
The hand that no one but his wife or
A keen-eyed druggist can decipher.

But though the doctor's script draws curses,
It's nothing, sometimes, to his nurse's—
A penmanship so full of scribbles
And blots and blurs and inky dribbles
That records get to be so heinous
You can't tell sinus, *oft, from* anus.

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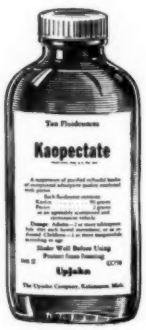


► DISEASES OF THE DIGESTIVE SYSTEM

Edited by Sidney A. Portis, M.D., Associate Clinical Professor of Medicine, University of Illinois Medical School, Chicago, Illinois. 57 contributors. Ed. 3, revised. 1,119 pages with 269 engravings and 5 color plates. 1953, Lea & Febiger, Philadelphia. $20.00.

Dr. Sidney Portis, and his pipe, have been familiar attendants of gastroenterologic assemblies for at least 2 decades. His cogent and pungent comments in discussion of the perennial topics, peptic ulcer and ulcerative colitis, have both enlightened and delighted a generation of admirers.

The first edition of "Diseases of the Digestive System" was published in 1941, with a distinguished corps of collaborators, with whom, however, the editor, Dr. Portis, did not refrain from taking issue, or even contradicting, in annotations. And this editorial practice enhanced the value of the work, as it does this third edition. The group of collaborators is formidable—the advance guard of gastroenterologists and allied specialists of America. It is to be hoped that the climate of California will not soften the trenchant attack on gastrointestinal problems that characterized Portis' Chicago attitude.

The slant toward psychogenic explanation of pathologic physiology of the gastrointestinal tract, indicated in the first edition and stressed in the second, is emphasized in the third edition. And this emphasis constitutes the chief merit of the presentation. For after all, the alimentary tract reflects, more than any other body system, psychoneurotic disturbances, and cognizance of this circumstance distinguishes the true gastroenterologic internist.

The physiology of the stomach, liver and biliary tract and of the pancreas is explained by Necheles, Ivy and Grossman, respectively. Quigley and Stewart Wolf adequately discuss the physiology of the small intestine and colon. Localization and interpretation of gastrointestinal tract pain are elucidated by Chester Jones.

Psychoneurotic aspects of gastrointestinal disturbance are discussed by Stanley, Cobb and Franz Alexander.

From this point the treatment of material is systemic, that is, esophageal, gastric, hepatic, biliary, enteric and colonic, each by a recognized authority in the particular field.

Actually, a compendium such as this cannot be reviewed, but mention of authors of sections such as Porter Vinson, esophagus; Schindler, gastritis; Palmer, peptic ulcer; Sara Jordan and Russell Boles, cancer of the stomach; Elman, pancreas; Wangensteen, appendix; Paulson, ulcerative colitis; Felsen, dysentery; Cohn, ileitis; Faust, parasitology, and many others of equal authority in their several fields indicates the scope of this work.

J. B. C.

Books received will be acknowledged in this department each month. As space permits, books of principal interest to our readers will be reviewed more extensively.

The Ciba Collection of Medical Illustrations. Volume 1: Nervous System (A Compilation of Pathological and Anatomical Paintings). By Frank H. Netter, M.D. 143 pages with 104 colored plates. 1953, Ciba Pharmaceutical Products, Inc., Summit, New Jersey. $6.00.

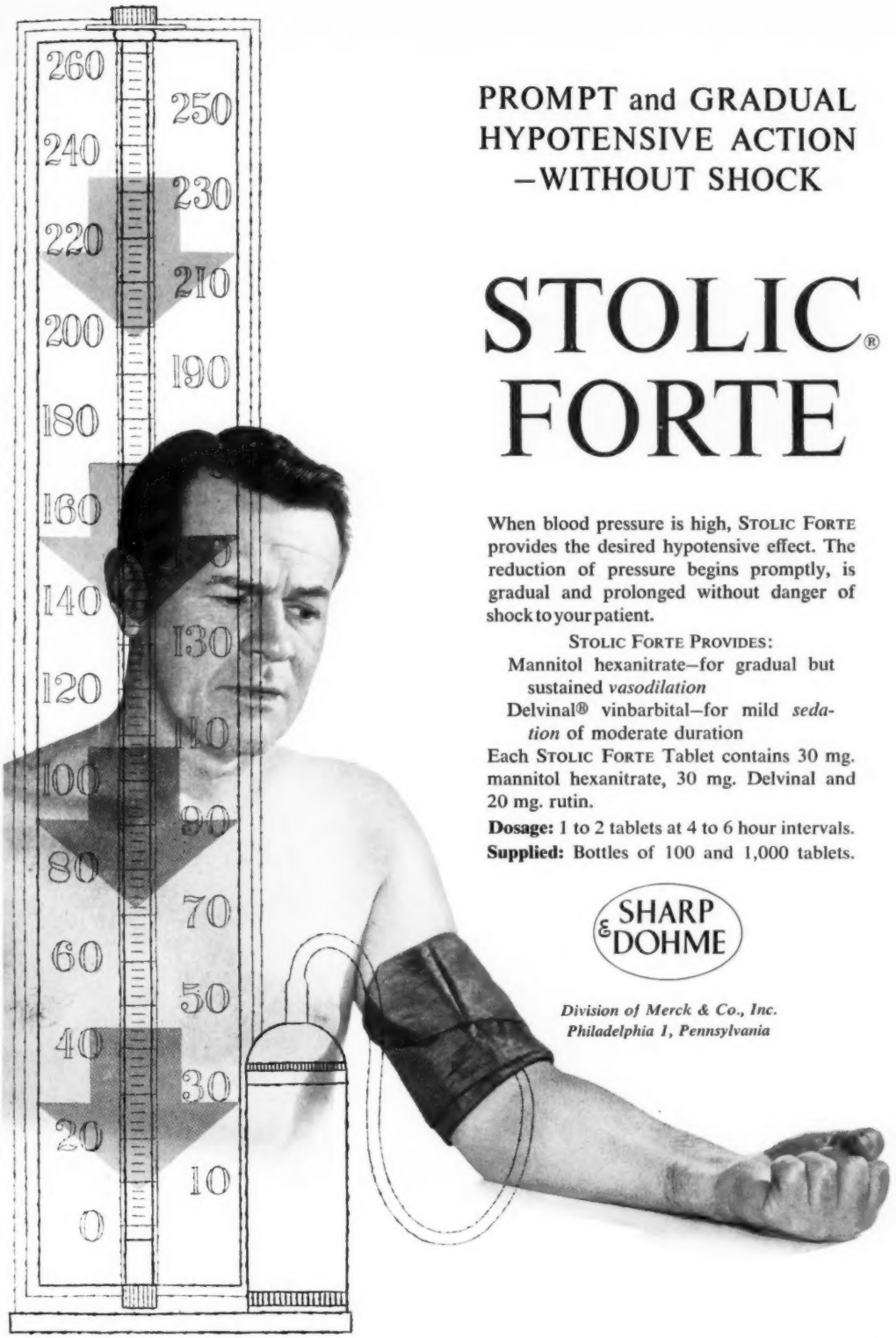
The Physician in Atomic Defense; Atomic Principles, Biologic Reaction and Organization for Medical Defense. By Thad P. Sears, M.D., Associate Clinical Professor of Medicine, University of Colorado School of Medicine. Foreword by James J. Waring, M.D., Professor of Medicine, University of Colorado School of Medicine. 308 pages. 1953, The Year Book Publishers, Inc., Chicago. $6.00.

The Epidemiology of Health. A New York Academy of Medicine Book. Edited by Iago Galdston, M.D. 197 pages. 1953, Health Education Council, New York. $4.00.

Tumors of the Orbit and Allied Pseudo Tumors: An Analysis of 216 Case Histories. By Raymond G. Ingalls, M.D., Former Instructor in Ophthalmology, College of Physicians and Surgeons, Columbia University, New York. 410 pages with 153 illustrations. 1953, Charles C Thomas, Springfield, Illinois. $11.50.

Conferences on Drug Addiction Among Adolescents. The New York Academy of Medicine, November 30, 1951 and March 13 and 14, 1952. Sponsored by the Committee on Public Health Relations of the New York Academy of Medicine, with the assistance of The Josiah Macy, Jr. Foundation. 320 pages. 1953, The Blakiston Company, New York & Toronto.

The Metabolism of the Tubercle Bacillus. By William F. Drea, D.M.D., Associate Laboratory Director, Colorado Foundation for Research in Tuberculosis, Colorado College, Colorado Springs, Colorado, and Anatole Andrejew, I.C., Research Staff, National Center for Scientific Research in France, Pasteur Institute, Paris. With a foreword by Esmond R. Long, M.D., Director and Professor of Pathology, University of Pennsylvania. 448 pages with 24 illustrations. 1953, Charles C Thomas, Springfield, Illinois. $12.50.



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